Hospital Library

ANNUAL MEETING NUMBER

The New England JOURNAL of MEDICINE

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL

Official Organ of The Massachusetts Medical Society
The New Hampshire Medical Society — The Vermont State Medical Society
The New England Surgical Society — The Boston Surgical Society, Inc.
The New England Pediatric Society — The New Hampshire Surgical Club
The New England Obstetrical and Gynecological Society
And The Neisserian Medical Society of Massachusetts

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CONTENTS

THE MASSACHUSETTS MEDICAL SOCIETY

Program of the One Hundred and Fifty-Third Anniversary 1043

NEW ENGLAND SURGICAL SOCIETY

Compression Fractures of Vertebral Bodies. *John P. Bowler* and *John F. Gile* 1052

The Necessary Requirements in a Minimum Diet for Infants and Children. *John Lovett Morse* 1057

The Use of Ergotamine Tartrate in Migraine. *William G. Lennox* 1061

The Parenteral Administration of Paraldehyde for the Control of Pain and Convulsive States. *Allen S. Johnson* 1065

Foreign Protein Sensitization with Meningeal Involvement Due to the Use of Vaccine. *William E. Hall* and *Thomas P. Murdock* 1067

Recurrent Dislocation of the Shoulder. *Frederic Jay Cotton* and *Gordon Mackay Morrison.* 1070

MEDICAL PROGRESS

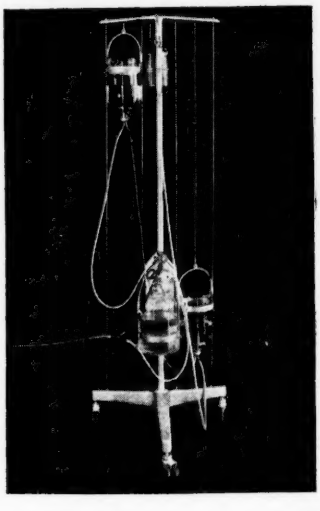
Progress in the Diagnosis and Treatment of Syphilis, 1933. *Austin W. Cheever* 1072

(Contents continued on next page)

THE NEXT ANNUAL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY WILL BE HELD IN WORCESTER, JUNE 4, 5 AND 6, 1934, AT THE HOTEL BANCROFT.

In Acute Bowel Obstruction

Due to Cancer of Rectum



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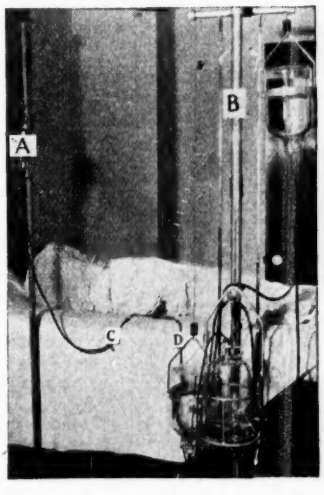

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CONTENTS

(Continued)

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 20201—Oliver Cope, Tracy B. Mallory, George A. Leland, Jr., William B. Breed, Fuller Albright and others. Case 20202—George W. Holmes, E. Parker Hayden, Robert R. Linton and Tracy B. Mallory..... 1077

EDITORIALS

The Massachusetts Medical Society. Distinguished Speakers for the Annual Meeting..... 1082
The Danger of Dinitrophenol as a Reducing Agent. 1082
The Tax Imposed by Tuberculosis..... 1083
Tribute to Dr. William Henry Welch..... 1083
This Week's Issue..... 1083

BOOK REVIEW

Aids to Neurology. E. A. Blake Pritchard..... 1094

BOOKS RECEIVED FOR REVIEW..... 1094

CORRESPONDENCE

Articles Accepted by the American Medical Association Council on Pharmacy and Chemistry... 1089
Free Lung Deflation Treatment. Walter C. Bailey and Bayard T. Crane..... 1089
May the Doctor Remove the Quarantine Card? M. J. Konikow..... 1089
Official Action of the Board of Registration in Medicine. Stephen Rushmore..... 1089
Tributes to the Memory of Dr. William Henry Welch. Stephen Rushmore. Arthur W. Allen. 1087
William Henry Welch. Anonymous..... 1088

MISCELLANY

Comparison of Disease Incidence in Connecticut with 1933 and Seven Year Average Month Ending April 28, 1934..... 1087
Massachusetts Department of Public Health. Division of Adult Hygiene. Cancer Clinic Bulletin. 1085
Mortality Rates..... 1076
One Way to Reduce Taxes. C.-E. A. Winslow..... 1084
Mr. Strawson Resigns..... 1076

NOTICE..... 1090

RECENT DEATHS..... 1090

REPORTS AND NOTICES OF MEETINGS..... 1091

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES..... 1094

The Massachusetts Medical Society

The next Annual Meeting of the Massachusetts Medical Society will be held in Worcester, at the Hotel Bancroft, Monday, Tuesday and Wednesday, June 4-5-6, 1934.

OFFICERS AND STANDING COMMITTEES ELECTED BY THE COUNCIL, JUNE 6, 1933

PRESIDENT: WILLIAM H. ROBEY, Boston.
VICE-PRESIDENT: PHILEMON E. TRUESDALE, Fall River.
SECRETARY: WALTER L. BURRAGE, Brookline.
TREASURER: CHARLES S. BUTLER, Boston.

STANDING COMMITTEES FOR 1933-1934

COMMITTEE OF ARRANGEMENTS
G. P. Reynolds, *Chairman;* W. M. Shedden, W. R. Morrison, Horatio Rogers, W. S. Burrage.

COMMITTEE ON PUBLICATIONS
R. I. Lee, *Chairman;* Homer Gage, R. B. Osgood, R. M. Smith, F. H. Lahey.

COMMITTEE ON MEMBERSHIP AND FINANCE
D. N. Blakely, *Chairman;* Gilman Osgood, G. C. Caner, J. E. Fish, H. F. Newton.

COMMITTEE ON ETHICS AND DISCIPLINE
David Cheever, *Chairman;* W. D. Ruston, S. F. McKeen, A. C. Smith, R. L. DeNormandie.

COMMITTEE ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS
Reginald Fitz, *Chairman;* C. H. Lawrence, C. A. Sparrow, E. S. Calderwood, A. S. Begg.

COMMITTEE ON STATE AND NATIONAL LEGISLATION
W. H. Robey, *Chairman;* T. J. O'Brien, F. E. Jones, A. W. Marsh, Shields Warren.

COMMITTEE ON PUBLIC HEALTH
Dwight O'Hara, *Chairman;* E. F. Cody, F. G. Curtis, Gerald Hoeffel, G. D. Henderson.

COMMITTEE ON MALPRACTICE DEFENSE
F. G. Balch, *Chairman;* E. D. Gardner, F. B. Sweet, R. P. Watkins, A. W. Allen.

COMMITTEE ON PERMANENT HOME: T. J. O'Brien, *Chairman;* S. B. Woodward, C. G. Mixter, J. M. Birnie, R. B. Greenough.

OFFICERS OF THE SECTIONS FOR 1934

ELECTED BY THE SECTIONS

(The street addresses may be obtained from the Annual Directory)

SECTION OF MEDICINE
Chairman, George R. Minot, Boston; *Secretary,* Herrman L. Blumgart, Boston.

SECTION OF SURGERY
Chairman, Ernest M. Daland, Boston; *Secretary,* Joseph H. Shortell, Boston.

SECTION OF TUBERCULOSIS
Chairman, Roy Morgan, Westfield; *Secretary,* Donald S. King, Boston.

SECTION OF PEDIATRICS
Chairman, Gardner C. Cobb, Worcester; *Secretary,* James M. Baty, Boston.

SECTION OF OBSTETRICS AND GYNECOLOGY
Chairman, J. W. O'Connor, Worcester; *Secretary,* Thomas Almy, Fall River.

SECTION OF RADIOLOGY AND PHYSIOTHERAPY
Chairman, Charles W. Blackett, Jr., Newtonville; *Secretary,* Franklin L. Lowry, Newton.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, BETWEEN APRIL 15 AND MAY 15, 1933.

(The street addresses may be obtained from the Annual Directory)

BARNSTABLE—*President,* J. L. Chute, Osterville; *Vice-President,* P. P. Henson, Hyannis; *Secretary,* J. I. B. Vail, Hyannis; *Treasurer,* H. B. Hart, Yarmouthport; *Librarian,* E. E. Hawes, Hyannis.

BERKSHIRE—*President,* M. H. Walker, Pittsfield; *Vice-President,* W. P. Kelly, Pittsfield; *Secretary,* H. J. Downey, Pittsfield; *Treasurer,* C. T. Leslie, Pittsfield.

BRISTOL NORTH—*President,* F. V. Murphy, Attleborough; *Vice-President,* L. E. Butler, Taunton; *Secretary,* C. B. Kingsbury, Taunton; *Treasurer,* J. V. Chatigny, Taunton.

BRISTOL SOUTH—*President,* J. H. Lindsey,* Fall River; *Vice-President,* F. M. Howes, New Bedford; *Secretary-Treasurer,* Charles Shanks, New Bedford.

ESSEX NORTH—*President* F. H. Coffin, Haverhill; *Vice-President,* John Parr, Methuen; *Secretary-Treasurer,* E. S. Bagnall, Groveland.

ESSEX SOUTH—*President,* R. E. Foss, Peabody; *Vice-President,* J. G. Corcoran, Hamilton; *Secretary,* R. E. Stone, Beverly; *Treasurer,* Andrew Nichols III, Danvers; *Librarian,* C. M. Cobb, Lynn.

FRANKLIN—*President,* A. E. Johnson, Greenfield; *Vice-President,* H. B. Marble, Shelburne Falls; *Secretary-Treasurer,* Charles Moline, Sunderland.

HAMPDEN—*President,* J. Z. Naurison, Springfield; *Vice-President,* A. J. Douglas, Westfield; *Secretary-Treasurer,* H. L. Smith, Springfield.

HAMPSHIRE—*President,* J. D. Collins, Northampton; *Vice-President,* T. F. Corriden, Northampton; *Secretary-Treasurer,* F. E. O'Brien, Haydenville; *Librarian,* Jane B. Armstrong, Northampton.

MIDDLESEX EAST—*President,* E. E. Tyzzer, Wakefield; *Vice-President,* J. H. Fay, Melrose; *Secretary,* A. R. Cunningham, Winchester; *Treasurer,* Richard Dutton, Wakefield; *Librarian,* J. M. Wilcox, Woburn.

MIDDLESEX NORTH—*President,* B. H. Pillsbury, Lowell; *Vice-President,* F. R. Mahony, Lowell; *Secretary,* T. A. Stamas, Lowell; *Treasurer,* M. D. Bryant, Lowell; *Librarian,* P. J. Meehan, Lowell.

MIDDLESEX SOUTH—*President,* A. H. Blake, West Somerville; *Vice-President,* S. H. Remick, Waltham; *Secretary,* A. A. Levi, Cambridge; *Treasurer,* Edward Mellus, Newton; *Librarian,* S. C. Dalrymple, Newton Lower Falls.

NORFOLK—*President,* A. S. Begg, West Roxbury; *Vice-President,* Cadis Phipps, Brookline; *Secretary,* F. S. Cruickshank, Brookline; *Treasurer,* G. W. Kaan, Sharon.

NORFOLK SOUTH—*President,* F. A. Bartlett, Quincy; *Vice-President,* T. B. Alexander, Scituate; *Secretary-Treasurer-Librarian,* N. R. Pillsbury, South Braintree.

PLYMOUTH—*President,* D. B. Tuholski, Brockton; *Vice-President,* L. A. Alley, Lakeville; *Secretary,* G. A. Moore, Brockton; *Treasurer,* A. C. Smith, Brockton; *Librarian,* J. H. Weller, State Farm (Bridgewater).

SUFFOLK—*Vice-President,* J. H. Means, Boston; *Secretary,* G. P. Reynolds, Boston; *Treasurer,* John Rock, Boston.

WORCESTER—*President,* W. C. Seelye, Worcester; *Vice-President,* E. B. Emerson, Rutland; *Secretary,* E. C. Miller, Worcester; *Treasurer,* E. P. Disbrow, Worcester; *Librarian,* A. C. Getchell, Worcester.

WORCESTER NORTH—*President,* C. E. Thompson, East Gardner; *Vice-President,* Lewis Fish,* Fitchburg; *Secretary,* F. M. McMurray, Fitchburg; *Treasurer,* F. H. Thompson, Jr., Fitchburg.

*Deceased.

Fellow members! We want your support.

The New England
Journal of Medicine

VOLUME 210 MAY 17, 1934 NUMBER 20

The Massachusetts Medical Society

PROGRAM OF THE ONE HUNDRED AND FIFTY-THIRD ANNIVERSARY

THE Exercises of the Anniversary will be held on Monday, Tuesday, and Wednesday June 4, 5, and 6 in the Hotel Bancroft and the adjoining Chamber of Commerce Building, Worcester.

WALTER L. BURRAGE, *Secretary.*
May 6, 1934.

1933 — 1934

OFFICERS OF THE SOCIETY

William H. Robey, *President*—202 Commonwealth Avenue, Boston.
Philemon E. Truesdale, *Vice-President* — 151 Rock Street, Fall River.
Walter L. Burrage, *Secretary* — 182 Walnut Street, Brookline.
Charles S. Butler, *Treasurer* — 257 Newbury Street, Boston.

George P. Reynolds, 311 Beacon Street, Boston —*Chairman, Committee of Arrangements.*
Edwin R. Leib, 36 Pleasant Street, Worcester —*Chairman, Local Committee of Arrangements.*

STANDING COMMITTEES

Of Arrangements

G. P. Reynolds, W. M. Shedden, W. R. Morrison, Horatio Rogers, W. S. Burrage.

On Publications

R. I. Lee, Homer Gage, R. B. Osgood, R. M. Smith, F. H. Lahey.

On Membership and Finance

D. N. Blakely, Gilman Osgood, G. C. Caner, J. E. Fish, H. F. Newton.

On Ethics and Discipline

David Cheever, W. D. Ruston, S. F. McKeen, A. C. Smith, R. L. DeNormandie.

On Medical Education and Medical Diplomas

Reginald Fitz, C. H. Lawrence, C. A. Sparrow, E. S. Calderwood, A. S. Begg.

On State and National Legislation

W. H. Robey, T. J. O'Brien, F. E. Jones, A. W. Marsh, Shields Warren.

On Public Health

Dwight O'Hara, E. F. Cody, F. G. Curtis, G. N. Hoeffel, G. D. Henderson.

On Malpractice Defense

F. G. Balch, E. D. Gardner, F. B. Sweet, R. P. Watkins, A. W. Allen.

On Permanent Home

T. J. O'Brien, S. B. Woodward, C. G. Mixter, J. M. Birnie, R. B. Greenough.

DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

Delegates

J. M. Birnie, Springfield; C. E. Mongan, Somerville; J. F. Burnham, Lawrence; W. H. Robey, Boston; E. F. Cody, New Bedford; R. I. Lee, Boston.

Alternates.

W. C. Leary, Springfield; L. S. McKittrick, Boston; E. L. Hunt, Worcester; Cadis Phipps, Brookline; P. E. Truesdale, Fall River; C. H. Lawrence, Jr., Boston.

GENERAL INFORMATION

Members of the medical profession are cordially invited to attend the exercises of the anniversary.

A Bureau of Information will be maintained by the Committee of Arrangements during the meetings in the lobby of the Hotel Bancroft.

Parking in the streets in the vicinity of the

hotel is restricted. Fellows are advised to use the parking spaces in the rear of the hotel reached by Portland and Federal Streets, or one of the three large garages which are within one or two minutes' walk.

Fellows are requested to register as soon as they arrive and to get their tickets to the Buffet Luncheon and the Annual Dinner on Tuesday, and the Buffet Luncheon on Wednesday. The charge for the Annual Dinner will be $2.00 to those whose dues have been paid.

On Tuesday, luncheon will be served *at a cover charge* to those *not members of the Council;* on Wednesday, *without charge* to those who are *not in arrears.*

The Worcester District Medical Society, at its Annual Meeting on Wednesday, May 9, very generously voted to assume the expense of entertainment for the Fellows and their wives. The Local Committee of Arrangements has arranged with the Worcester, Tatnuck and Wachusett Country Clubs to permit us to play golf on their links at any time during the three days of the meeting. The District Society has also arranged entertainment for the ladies on June 4 and 5. Under the care of a committee of ladies of the Worcester District Society visiting ladies will be taken in busses to the chief points of interest in the city and vicinity, including the new Auditorium, the Higgins Armory, where a very remarkable collection of armor is on exhibition, the Worcester Art Museum, where many fine works of art are on display, and some of the beautiful gardens in and about Worcester. They will also be entertained at luncheons, both Monday and Tuesday, at the Worcester Country Club. Full details will be furnished by the Chairman of the Local Committee of Arrangements, Dr. Edwin R. Leib, 36 Pleasant street, Worcester. Fellows are cordially invited to bring their wives to Worcester.

The Scientific Exhibits will be in the small ballroom immediately back of the main ballroom of the Hotel Bancroft and also on the mezzanine floor above the ballroom and the lobby. See page 1047 for list of exhibits.

The Commercial Exhibits will occupy the lobby of the hotel, the dining room, and the ballroom assembly immediately off the main ballroom. A list of the exhibitors will be found on page 1049.

MONDAY MORNING, JUNE 4
9:30 TO 12:30 O'CLOCK

There will be dry clinics at the following hospitals: Belmont Hospital, Belmont Street; Fairlawn Hospital, 189 May Street; Memorial Hospital, 119 Belmont Street; St. Vincent Hospital, 73 Vernon Street; Worcester City Hospital, 71 Jacques Avenue; Worcester Hahnemann Hospital, 281 Lincoln Street; Worcester State Hospital, Belmont Street.

CLINIC PROGRAMS

MEMORIAL HOSPITAL—JUNE 4, 1934
10:00 A.M.-12:30 P.M.

10:00-11:00—*Obstetrical Service*
Single Blade Forceps Rotation—Moving Pictures.
Abruptio Placentae—Case Reports with lantern slides.
Drs. G. C. Lincoln, J. W. O'Connor, J. E. Talbot.

Medical Service
Ward Rounds.
Demonstration of Unusual Cardiac Cases with Electrocardiograms.
Drs. O. H. Stansfield and E. J. Halloran.

10:30-12:30—*Pediatrics Service*
(1) Demonstration of Interesting Cases of Blood Dyscrasia
(2) Mongolism in Twins—case
(3) Muscular Dystrophy—cases
(4) Children's Diabetic Manual
(5) Pasteurization—efficiency
(6) Modified Home Oxygen Tent
(7) Portable Cubicles
Drs. C. A. Sparrow, A. M. Kimberly, H. Talamo.

11:00-12:00—*Radiology*
Treatment of Skin Cancer—Cases and lantern slides.
Dr. R. Dresser.

11:00-12:30—*Nose and Throat Service*
Bronchoscopy.
Cleft Palate.
Tonsil Operative Technique.
Drs. Gordon Berry, H. J. Gibby, A. L. Caron, H. B. Goodspeed.

Surgical Service
Varicose Veins—Saphenous Ligation.
Abdominal Menstrual Fistula—Case Operations.
Drs. W. C. Seelye, B. H. Alton, D. S. Adams, C. T. Smith, B. C. Wheeler.

12:30—Luncheon.

WORCESTER CITY HOSPITAL
JUNE 4, 1934
10:00 A.M.-12:30 P.M.

10:00-12:00 A.M.—Winslow Surgery—Surgical Clinic. Dr. B. F. Andrews and members of Surgical Staff.
Participating in the Clinic: Dr. F. H. Lahey and associates presenting brain surgery, surgery of the colon and surgery of the gallbladder.

10:00 A. M.—Thayer Hall—Medical Clinic
Amoebic Dysentery. Presentation by Dr. Percy A. Brooke.
Poliomyelitis. Presentation by Dr. James A. Givan.
Diseases of the Blood. Presentation by Dr. Raymond H. Goodale.

Heart Disease. Presentation by Dr. Frank B. Carr.

Diabetes. Presentation by Dr. A. Wilson Atwood.

Participating in the Clinics: Dr. Elliott P. Joslin, Boston; Dr. Burton E. Hamilton, Boston; Dr. William Dameshek, Boston; and others.

12:30 P.M.—Thayer Hall—Luncheon

Details for the other hospital clinics will be announced later.

The luncheons at the various hospitals, following these clinics, are to be served through the courtesy of the Worcester District Medical Society.

MONDAY AFTERNOON, JUNE 4

2:30 O'Clock

Hotel Bancroft, Crystal Room

Section of Pediatrics

Officers of the Section

Dr. Gardner N. Cobb, Worcester, *Chairman.*
Dr. James M. Baty, Boston, *Secretary.*

1. *The Use of the Ketogenic Diet in Pyuria.*
   By Dr. Warren E. Wheeler, Massachusetts General Hospital, Boston. (By invitation.)
   Discussion to be opened by Dr. Fritz B. Talbot, Boston.
2. *Present Status of Vaccination Against Tuberculosis.*
   By Dr. Clement A. Smith, Children's Hospital, Boston. (By invitation.)
   Discussion to be opened by Dr. Eliot H. Luther, Westfield, and Dr. Arnold Branch, Boston. (By invitation.)
3. *The Rôle of the Streptococcus in So-called Sudden Death in Infancy and Childhood.*
   By Dr. Sidney Farber, Harvard Medical School, Boston. (By invitation.)
   Discussion to be opened by Dr. Richard M. Smith, Boston, and Dr. Karlton G. Percy, Chestnut Hill and Boston.
4. *Pyogenic Infection of Bones and Joints in Infants.*
   By Dr. William T. Green, Children's Hospital, Boston.
   Discussion to be opened by Dr. Frank R. Ober, Boston, and Dr. Henry W. Hudson, Jr., Waban and Boston.

MONDAY AFTERNOON, JUNE 4

2:30 O'Clock

Hotel Bancroft, Ballroom

Section of Radiology and Physiotherapy

Officers of the Section

Dr. Charles W. Blackett, Newtonville and Boston, *Chairman.*
Dr. Franklin P. Lowry, Newton, *Secretary.*

1. *A Method of Reduction and Fixation of Intracapsular Fractures of the Femoral Neck.*
   By Dr. Joseph H. Fay, Melrose.
   Discussion to be opened by Dr. Ralph D. Leonard, Melrose and Boston.
2. *The Conduct of the Convalescent Stage of Anterior Poliomyelitis.*
   Motion pictures showing a new method of muscle re-education.
   By Dr. Harold D. Corbusier, Plainfield, New Jersey. (By invitation.)
   Discussion to be opened by Dr. Frank R. Ober, Boston.
3. *Diverticulosis, Diverticulitis and Carcinoma of the Colon: A Roentgenologic Discussion.*
   By Dr. Ross Golden, New York, N. Y. (By invitation.)
   Discussion to be opened by Dr. Richard B. Cattell, Boston; Dr. Merrill C. Sosman, Boston; Dr. Philip H. Cook, Worcester.
4. *Physical Therapy in the Treatment of Athletic Injuries.*
   By Dr. Thomas K. Richards, Boston.
   Discussion to be opened by Dr. Otto J. Hermann, Brookline and Boston.

MONDAY EVENING, JUNE 4

8 O'Clock

Hotel Bancroft, Ballroom

The Shattuck Lecture

By Dr. Warfield T. Longcope, Baltimore, Physician-in-Chief, The Johns Hopkins Hospital.

Subject: *The Importance of Disturbances in Nutrition in Oedematous States.* (Illustrated by lantern slides.)

Light refreshments will be served after the lecture.

TUESDAY MORNING, JUNE 5

9:15 O'Clock

Chamber of Commerce Hall

Section of Surgery

Officers of the Section

Dr. Ernest M. Daland, Boston, *Chairman.*
Dr. Joseph H. Shortell, Boston, *Secretary.*

1. *Cancer of Rectum and Lower Bowel. Case Problems and Methods.*
   By Dr. Ernest L. Hunt, Consulting Surgeon, Worcester City Hospital.
   Discussion to be opened by Dr. F. B. Sweet, Springfield, and Dr. Benjamin H. Alton, Worcester.
2. *Total Ablation of Thyroid Gland for Intractable Heart Disease. Medical and Surgical Aspects.*

By Dr. Herrman L. Blumgart, Boston, Associate Professor of Medicine, Harvard Medical School, and Visiting Physician, Beth Israel Hospital, and Dr. David D. Berlin, Boston, Assistant Professor of Anatomy, Tufts College Medical School, and Surgeon to Thyroid Clinic, Beth Israel Hospital.

Discussion to be opened by Dr. Howard M. Clute of the Lahey Clinic, Boston, and Dr. Robert C. Cochrane, Surgeon-in-Chief, Second Surgical Service, Boston City Hospital.

3. *Trans-Urethral Resection of the Prostate.*

By Dr. Herman C. Bumpus, Jr., New York. Recently at the Mayo Clinic, Rochester, Minnesota. (By invitation.)

Discussion to be opened by Dr. Roger C. Graves, Visiting Urologist, Carney Hospital, and Genito-Urinary Surgeon, Pondville Hospital, and Dr. Walter D. Bieberbach, Worcester, Urologist to Worcester City Hospital.

4. *Management of Fractures About the Ankle Joint.*

By Dr. Clay Ray Murray, New York, Assistant Professor of Surgery, College of Physicians and Surgeons, Columbia University. (By invitation.)

Discussion to be opened by Dr. Frederic J. Cotton, Boston, Consulting Surgeon, Boston City Hospital, and Dr. Charles L. Scudder, Boston, Honorary Surgeon, Massachusetts General Hospital.

TUESDAY MORNING, JUNE 5

11:30 O'Clock

Hotel Bancroft, Ballroom

ANNUAL MEETING OF THE SUPERVISING CENSORS

TUESDAY NOON

Hotel Bancroft, Ballroom

ANNUAL MEETING OF THE COUNCIL

Followed by the Cotting Luncheon to Councilors in the Chamber of Commerce Hall.

Other stated meetings in John Ware Hall, Boston Medical Library, 8 Fenway, at noon, on the first Wednesdays of October and February.

TUESDAY AFTERNOON, JUNE 5

1 O'Clock

Chamber of Commerce Hall

A buffet luncheon will be served to Fellows who have tickets. (Procurable at Registration Desk. Price $1.00.)

2:30 O'Clock

Chamber of Commerce Hall

SECTION OF MEDICINE

Officers of the Section

Dr. George R. Minot, Boston, *Chairman.*
Dr. Herrman L. Blumgart, Boston, *Secretary.*

1. *The Social Conditioning of the Visceral Activities.*

By Dr. Abraham Myerson, Boston, Professor of Neurology, Tufts College Medical School; Director of Research, Boston State Hospital; Senior Visiting Neurologist, Boston City Hospital and Beth Israel Hospital.

Discussion to be opened by Dr. Lawrence K. Lunt, Carlisle and Concord.

2. *Cancer and the General Practitioner.*

By Dr. Henry Jackson, Jr., Chestnut Hill and Boston, Assistant Professor of Medicine, Harvard Medical School; Associate Physician, Thorndike Memorial Laboratory; and Junior Visiting Physician, Boston City Hospital; Physician, Pondville Hospital.

Discussion to be opened by Dr. P. E. Truesdale, Fall River.

3. *Chronicity of Rheumatic Fever.*

By Dr. Homer F. Swift, New York, Member of the Rockefeller Institute for Medical Research. (By invitation.)

Discussion to be opened by Dr. Paul D. White, Boston, and Dr. Oliver H. Stansfield, Worcester.

4. *Tuberculosis as It Affects the Physician's Practice.*

By Dr. Henry D. Chadwick, Boston, Massachusetts Commissioner of Public Health.

Discussion to be opened by Dr. Brace W. Paddock, Pittsfield.

TUESDAY EVENING, JUNE 5

7 O'Clock

Hotel Bancroft, Ballroom

THE ANNUAL DINNER

Fellows wishing to sit together at the dinner will please send their names to Dr. Edwin R. Leib, *Chairman of the Local Committee of Arrangements,* 36 Pleasant Street, Worcester, at the earliest possible date and proper reservations will be made. Tickets for the dinner should be obtained at the Registration Desk. The galleries of the ballroom will be reserved for ladies who wish to hear the distinguished after-dinner speakers.

WEDNESDAY MORNING, JUNE 6

9 O'Clock

Chamber of Commerce Hall

SECTION OF OBSTETRICS AND GYNECOLOGY

Officers of the Section

Dr. Joseph W. O'Connor, Worcester, *Chairman.*
Dr. Thomas Almy, Fall River, *Secretary.*

1. *Prolan Therapy in Abnormal Uterine Bleeding.* (Illustrated by lantern slides.)
   By Dr. Joe Vincent Meigs, Boston, Gynecologist.
   Discussion to be opened by Dr. Frank A. Pemberton, Boston, Gynecologist and Dr. Charles H. Lawrence, Jr., Boston, Internist.
2. *Intracranial Hemorrhage in the New Born from the Standpoint of the General Practitioner.* (Illustrated by lantern slides.)
   By Dr. P. Brooke Bland, Professor of Obstetrics, Jefferson Medical College, Philadelphia. (By invitation.)
   Discussion to be opened by Dr. Alonzo K. Paine, Boston, Obstetrician, Dr. Richard S. Benner, Springfield, Obstetrician, and Dr. Randolph K. Byers, Milton and Boston, Pediatrician.
3. *Radium Therapy in Uterine Hemorrhages of Benign Origin—An Analysis of the End-Results of 150 Consecutive Cases.* (Illustrated by lantern slides.)
   By Dr. Louis E. Phaneuf, Boston, Gynecologist.
   Discussion to be opened by Dr. Samuel R. Meaker, Boston, Gynecologist, and Dr. Richard Dresser, Chestnut Hill and Boston, Radiologist.

WEDNESDAY MORNING, JUNE 6

9 O'Clock

Hotel Bancroft, Crystal Room

SECTION OF TUBERCULOSIS

Officers of the Section

Dr. Roy Morgan, Westfield, *Chairman.*
Dr. Donald S. King, Boston, *Secretary.*

Symposium on Measurement of Activity in Pulmonary Tuberculosis. (Papers limited to eight minutes each.)

1. *Symptoms.*
   Dr. Harvey S. Wagner, Barnstable County Sanatorium, Pocasset.
2. *Physical Signs.*
   Dr. Frank H. Hunt, Boston Sanatorium, Mattapan.
3. *X-ray.*
   Dr. Olin S. Pettingill, Essex County Sanatorium, Middleton.
4. *Quantitative Tuberculin Tests.*
   Dr. Theodore L. Badger, Boston City Hospital.
5. *Leucocyte Picture.*
   Dr. Jacob Kaminsky, Middlesex County Sanatorium, Waltham.
6. *The Monocyte-Lymphocyte Ratio.*
   Dr. Gulli Lindh Muller, Rutland State Sanatorium.
7. *Schilling Differential Count and Red Cell Sedimentation Rate.*
   Dr. John W. Cass, Jr., The Channing Home, Boston.
8. *Discussion and Summary of the Whole Problem with Especial Reference to the Study of the Leucocyte Count in Pulmonary Tuberculosis.*
   Dr. William H. Ordway, Physician-in-Charge, Metropolitan Life Insurance Company Sanatorium, Mt. McGregor, New York State. (By invitation.)

WEDNESDAY NOON, JUNE 6

Hotel Bancroft, Ballroom

ANNUAL MEETING OF THE SOCIETY

BUSINESS OF THE ANNUAL MEETING

A draft, second revision, of the By-Laws has been sent to every Fellow, under the terms of Chapter VI, Section 3, and Chapter IX of the By-Laws.

WEDNESDAY AFTERNOON, JUNE 6

1 O'Clock

Hotel Bancroft, Ballroom

THE ANNUAL DISCOURSE

By Dr. Lincoln Davis, Boston, Consulting Surgeon, Massachusetts General Hospital; President, Boston Medical Library.

Subject: *The Objectives of Medical Progress.*

WEDNESDAY AFTERNOON, JUNE 6

2 O'Clock

Chamber of Commerce Hall

Buffet Luncheon to all with tickets.

This will end the Annual Meeting.

SCIENTIFIC EXHIBITS

1. *Some Effects of Liver Extracts Injected Intramuscularly.* By Dr. William Parry Murphy, Boston, Instructor in Medicine, Harvard Medical School. Illustrated by charts and moving pictures.
2. *Postgraduate Medical Instruction in Massachusetts.* By the Massachusetts Medical Society, Committee on Postgraduate Medical Instruction. Dr. Leroy E. Par-

kins, Boston, Secretary. The past year's experience in postgraduate instruction will be shown in graphic form, together with an outline of next year's program.

3. *Poliomyelitis—its pathology, epidemiology and after-care.* By Dr. A. T. Legg, Boston, Assistant Professor of Orthopedic Surgery, Harvard Medical School; Dr. W. L. Aycock, Boston, Director of Research, Harvard Infantile Paralysis Commission; and Janet B. Merrill, Boston, Director of Physical Therapeutics, Harvard Infantile Paralysis Commission and Children's Hospital. In addition to statistical charts and models, a moving picture will be shown to illustrate the management of a case from the beginning of the orthopedic treatment.

4. *The Modern Conception of Gonorrhea.* By the American Social Hygiene Association and the Neisserian Medical Society of Massachusetts. Dr. M. J. Exner, New York, N. Y. (By invitation.) There will be charts illustrating all the phases of diagnosis and treatment. This material has been prepared by Dr. Edward L. Keyes, Dr. Emily D. Barringer, Dr. Erick Langer, Dr. P. S. Pelouze, Dr. Anson Clark and others. The exhibit won first honors at the American Medical Association meeting in 1933.

5. *The Control of Diabetes.* By the Metropolitan Life Insurance Company. Dr. Louis I. Dublin, third vice-president and statistician, and the George F. Baker Clinic of the New England Deaconess Hospital, Dr. Elliott P. Joslin, Boston, Medical Director. This will be a revision and enlargement of the exhibit shown at the American Medical Association meeting, illustrated by charts and lantern slides.

6. *Treatment of Intractable Congestive Failure and Angina Pectoris by Total Ablation of the Normal Thyroid Gland.* By Dr. H. L. Blumgart, Boston, Associate Professor of Medicine, Harvard Medical School. There will be charts demonstrating the technique of treatment, the therapeutic results, biochemical and electrocardiographic studies, and moving pictures of the various aspects of thyroidectomy. The exhibit will represent the work of Dr. Blumgart, Dr. A. A. Weinstein, Dr. David Davis, Dr. J. E. F. Riseman, and Dr. D. D. Berlin.

7. *Fungi Pathogenic to Man.* By Dr. John G. Downing, Newton and Boston, Assistant Professor of Dermatology, Tufts College Medical School; W. H. Weston, Professor of Cryptogamic Botany, Harvard University; and Sarah M. Cousins, Harvard School of Biology. (By invitation.) The organisms from the simplest forms to the most complex pathogens will be shown growing in culture, together with microphotographic enlargements of their appearance in culture and in tissue lesions, and photographs of the characteristic lesions produced.

8. *Lobar Pneumonia, Its Treatment, and the Neufeld Method of Pneumococcus Typing.* By the Massachusetts Department of Public Health, Division of Communicable Diseases; Dr. G. W. Anderson, Waban and Boston, Director, and Dr. Roderick Heffron, Boston, Director of Pneumonic Study. In addition to the graphic presentation of the results of the recent special pneumonia study in this state, there will be demonstrations of the technique of rapid pneumococcus typing.

9. *Arthritis.* By the Massachusetts Department of Public Health, Division of Adult Hygiene, Dr. H. L. Lombard, Newton Centre and Boston, Director. There will be gross specimens demonstrating the various types of arthritic pathology, with statistical charts prepared by the Division of Adult Hygiene.

10. *Cancer.* By the Massachusetts Department of Public Health, Dr. G. W. Taylor, Brookline and Boston, Surgeon at Pondville Hospital; Dr. Shields Warren, Boston, Pathologist to Pondville Hospital; and Dr. H. L. Lombard, Newton Centre and Boston, Director, Division of Adult Hygiene. Lantern slides, photographs and x-rays of characteristic lesions showing results of various therapeutic measures, and statistical charts of the incidence, curability and mortality rates of cancer in its several forms.

11. *Silicosis and Tuberculosis in Granite and Foundry Workers.* By the Massachusetts Department of Public Health, Division of Tuberculosis, Dr. A. S. Pope, Newtonville and Boston, Director. The results of the study made by the special Industrial Disease Commission of 1933.

12. *Social Aspects of Medicine.* By a Committee of the Harvard Medical School on the Social Aspects of Medicine, and the Chiefs of the Social Service Department of the Boston City Hospital, the Massachusetts General Hospital, the Beth Israel Hospital, and the Children's Hospital. This exhibit illustrates the value of an understanding of the social aspects of medicine to diagnosis, prognosis, therapeusis and prevention of disease.

Censors' Meetings

The Censors for the several districts will meet for the examination of applicants for fellow-

ship on the first Thursdays of May and November.

The Censors for the Suffolk District will examine applicants residing in that district and also applicants who are non-residents of Massachusetts.

Applicants for fellowship should apply to the Secretary of the District Society of the district in which they reside (have a legal residence) at least two weeks before the date of a given examination, taking with them their diplomas.

Treasurer's Notice

Assessments, payable in advance, should be paid to the District Treasurers, or, in the case of non-residents, to the Treasurer.

Assessments were due January first. For the convenience of Fellows who have not yet paid, such assessments will be received for the Treasurer at the Registration Desk in the lobby of the hotel.

Secretary's Notice

All communications as to membership, especially changes of residence and address, should be sent to the Secretary, who keeps a constantly corrected official list of the Fellows and their addresses.

The Journal

The *New England Journal of Medicine*, the official weekly organ of the Society, will be sent only to Fellows who have paid their assessments, and to such Retired Fellows as may apply for it. Address communications to the Managing Editor of the *Journal*, Dr. Walter P. Bowers, 8 Fenway, Boston.

Society Headquarters,
8 Fenway, Boston

COMMERCIAL EXHIBITS

Booth No.

1—The P. L. Rider Company, 9 Norwich Street, Worcester, Massachusetts.

The P. L. Rider Company, jobbers of general hospital supplies, will display a complete line of Instruments and Specialties. L. H. Marsden of Worcester will be in charge.

2—The Medical Protective Company, Wheaton, Illinois.

Provides professional protection exclusively. A. F. Fox and F. G. Lockwood, in charge of the exhibit, will be at your service.

3—Tailby-Nason Company, Boston, Massachusetts.

Manufacturers of Nason's Palatable Cod Liver Oil. The Giant Cod and photographs of the Lofoten Fisheries in Norway will be an interesting part of the exhibit.

4—The Kellogg Company, Battle Creek, Michigan.

Manufacturers of ready-to-eat cereals and Kaffee Hag Coffee. Kaffee Hag Coffee will be served and all delegates are invited to call at the booth, have a sample and see the caffeine exhibit. Mary I. Barber, Director of Home Economics, will be in charge.

5—General Electric X-Ray Corporation, 624 Beacon Street, Boston, Massachusetts.

Manufacturers of x-ray and electro-medical apparatus. Shock-proof x-ray apparatus, x-ray films, an Inductotherm and minor equipment will be displayed. J. W. Moulton of Springfield will be in charge.

6—The Brookhill Laboratories, Genesee Depot, Wisconsin.

A new method of preparing Concentrated Acidophilus Milk is to be introduced. R. E. Sampson of the Brookhill Agency, Boston, will be present to explain details of manufacturing processes.

7—Kilham & Maleod, 56 Central Avenue, Lynn, Massachusetts.

Exclusive New England distributers for Lepel High Frequency Laboratories of New York. A complete line of physical therapy equipment will be displayed, showing the latest development notably the new combination quartz ultra-violet lamps and cold ultra-violet lamps, a high powered diathermy equipment in one combination unit; also the latest miniature quartz ultra-violet lamps for internal use. An all purpose electrode surgical unit capable of doing all types of electrode surgery including transurethral prostatic resection and combination short wave diathermy and electrode surgical units.

8—Westinghouse X-Ray Company, Inc., Long Island City, New York.

Manufacturers of electro-medical apparatus. A new line of our x-ray accessories together with the Westinghouse new Model "G" Endotherm will be displayed.

9—Lederle Laboratories, Inc., 511 Fifth Avenue, New York, New York.

This exhibit will present the more important Biological and Pharmaceutical Products which they manufacture. Among the Biological Products which will be shown are Pollen Antigens, which for twenty years have proved successful in controlling attacks of hay fever; Poison Ivy Extract in almond oil for Rhus Dermatitis; Antipneumococcic Serum for pneumonia; the single dose Toxoid for diphtheria prevention; Tetanus-Gas-Gangrene Antitoxin for prophylaxis; and Staphylococcus Toxoid for the prevention and control of staphylococcic infections.

Their pharmaceuticals include Solution Liver Extract Parenteral for pernicious anemia; Solution Liver Extract Oral; Ferric Ammonium Citrate in capsules; Cod Liver Oil Concentrate Tablets and standardized Whole Leaf Digitalis Tablets.

10—Winthrop Chemical Company, Inc., and H. A. Metz Laboratories, Inc., 170 Varick Street, New York, New York.

At the Winthrop-Metz booth you will find a special exhibit of some of the newest products

of this firm. Special attention is called to Novaldin, the analgesic with a wide safety quotient; Myorgal, vasodilator for angina; Synodal, for injection therapy of peptic ulcer; Diodrast, radiopaque agent for intravenous urography. Literature gladly supplied on these, as well as Curtasal, Avertin, Luminal, Theominal, Phanodorn, Pyramidon, Novocain, Salyrgan, Omnadin and the Salvarsans.

11—Gerber Products Company, Fremont, Michigan.

Any information desired concerning the Gerber's Strained Cereal, Vegetables, and Prunes will be gladly given. Leaflets and booklets are available for use in your practice or for your own information. Gerber Products are used both for infant feeding and in therapeutic diets and literature along those lines will be supplied.

12—Petrolagar Laboratories, 8134 McCormick Blvd., Chicago, Illinois.

Each tablespoonful of this new type of Petrolagar contains the physiological equivalent of the U. S. P. dose (2 cc.) Fluid extract Cascara Sagrada U. S. P. There is a sample of Petrolagar with Cascara for every visitor.

13—Massachusetts State Pharmaceutical Association, 20 Glen Road, Newton Centre, Massachusetts.

A line of U. S. Pharmacopoeial and National Formulary preparations will be displayed.

14—R. B. Davis Company, Hoboken, New Jersey.

Cocomalt, the scientific food concentrate that supplies a rich calcium, phosphorus and Vitamin D content in a delicious form, is again being exhibited. Physicians report an ever-increasing use of Cocomalt in the diet. Interesting scientific data available. Scientific information will be given, and questions on Cocomalt will be answered.

15, 16—Merck & Co., Inc., Rahway, New Jersey.

Manufacturers of fine prescription chemicals. In addition to such well-known therapeutic agents as Tryparsamide, Bismosol, Stovarsol, and Pyridium, several interesting new preparations such as Mecholin, for the treatment of paroxysmal tachycardia and peripheral vascular diseases will be exhibited.

17—Physician-Dentist Service, Inc., 224-225 Park Square Building, Boston, Massachusetts.

Finances the patient for the doctor or hospital. A complete explanation of their plan will be available. J. R. Watson, Assistant-Treasurer, will be in charge.

18—The DeVilbiss Company, Toledo, Ohio.

Manufacturers of medicinal atomizers and vaporizers. A complete line of medicinal atomizers and vaporizers will be on exhibit. A prominent feature of this display will be the new DeVilbiss Nasal Guard, which eliminates any excess pressure in the nasal passages during prescribed self-treatment with an atomizer. E. Manning of the Boston Office will be in charge.

19—Crosbie-Macdonald Insurance Agency, 79 Milk Street, Boston, Massachusetts.

Specializing in protection for members of the Massachusetts Medical Society, against reputation, good name, working ability, instruments, your life and retirement can be provided for. They will be glad to help solve your problems.

20—Moore & Company, Inc., Worcester, Massachusetts.

21—S. M. A. Corporation, Cleveland, Ohio.

22—Hynson, Westcott & Dunning, Charles and Chase Streets, Baltimore, Maryland.

23—Campbell X-Ray Co. of Boston, 100 Sudbury Street, Boston, Massachusetts.

X-ray and electro-medical apparatus made in Boston will be displayed.

24—Mead Johnson & Company, Evansville, Indiana.

A complete line of infant diet materials including Dextri-Maltose, Mead's Newfoundland Cod Liver Oil, Mead's Viosterol in Oil 250 D, Mead's 10 D Cod Liver Oil, Mead's (A-D) Viosterol in Halibut Liver Oil 250 D (Liquid and Capsules), Mead's Halibut Liver Oil, Mead's Brewers Yeast Powder, Mead's Brewers Yeast Tablets, Pablum, Mead's Cereal, Sobee, Mead's Powdered Protein Milk, Mead's Powdered Lactic Acid Milk, Powdered Whole Milk, Alacta, Recolac and Casco will be displayed.

26—The Mellin's Food Company, 177 State Street, Boston, Massachusetts. Manufacturers of Mellin's Food.

27—Charles W. Broadbent Company, 665 Huntington Avenue, Boston, Massachusetts.

A complete line of Medical, Surgical and Hospital Supplies will be displayed. Charles W. Broadbent will be in charge.

28, 29—E. F. Mahady Company, 851-857 Boylston Street, Boston, Massachusetts.

The new Burdick SU2 Endo-thermic Knife utilizing a blended current comprising patented cutting and coagulating features will be displayed. Also the new Baxter's Intravenous Solutions, the Scialytic Flash emergency and operating light, and many other new items of interest to the medical profession.

30—Horlick's Malted Milk Corporation, Racine, Wisconsin.

Manufacturers of Horlick's the Original Malted Milk. A complete line of Horlick's the Original Malted Milk, powder and tablet forms, natural and chocolate flavors will be displayed. Dr. J. A. Denkinger of Boston will be in charge.

31—Association of Certified Milk Producers of Metropolitan Boston, 1106 Boylston Street, Boston, Massachusetts.

The methods and technical control procedures pursued in the production of Certified Milk will be presented. Certified Milk is produced under medical supervision to fulfill the most exacting requirements of the medical profession for clean, safe milk of optimum nutritional values.

32—Davies, Rose & Co., Ltd., 22 Thayer Street, Boston, Massachusetts.

While Pil. Digitalis (Davies, Rose) needs no introduction, there are, perhaps, those who are

not familiar with this firm's other preparations. Messrs. Mansfield and Fleming will be in charge.

33—The E. L. Patch Company, Stoneham Post Office, Boston, Massachusetts.

The various Patch specialties will be displayed.

Booths A and B—The Stetson Shoe Company, South Weymouth, Massachusetts.

Manufacturers of high-grade shoes for men and women. A complete line of Stetson Shoes will be displayed. C. C. Mason will be in charge.

Booth C—The J. B. Lippincott Company, Philadelphia, Pennsylvania.

The J. B. Lippincott Company will exhibit an unusual line of new and standard medical, surgical, pharmaceutical and nursing books. Among the most outstanding is Peham and Amreich—"Operative Gynecology," in two volumes. This atlas is beautifully and extensively illustrated by large drawings showing each operation step by step. Practically all of them are beautifully and accurately colored.

Kirschner—"Operative Surgery," in two volumes. This book immediately became known as "The Color Surgery" because of the wealth of detailed colored illustrations. It contains a great number of items that cannot be found in any other Operative Surgery and is well worth the most careful scrutiny.

There is a new edition of the well-known doctor's time saver, Lippincott's Quick Reference Book and an entirely new and most inexpensive work on "The Treatment of the Commoner Diseases" by Lewellys F. Barker, and the new idea in personal post-graduate work instruction at home supplied from the Pittsburgh Diagnostic Clinic as a Supplement to the famous International Clinics.

In the nursing field, there is an entirely new work by Solomon on "Pharmacology, Materia Medica and Therapeutics for Nurses" and a number of new editions of the standard nursing text.

TUFTS ALUMNI LUNCHEON

A Tufts Medical Alumni luncheon is scheduled for Monday, June 4th, at 12:30 P.M. at the Hotel Bancroft, Worcester.

Careful perusal by the Fellows of the Society of the program set forth should arouse that degree of enthusiasm which would prompt designation in one's engagement book of the three days of the Annual Meeting. Every section is well arranged to present important educational material, and the accepted estimate of a practitioner is based on his attendance at medical meetings.

Last year the scientific exhibit was unanimously approved as a very interesting and instructive feature of the session. That arranged for the Worcester meeting presents significant advancements in medicine. The commercial exhibits should interest every doctor, for the best pharmaceutical skill is shown in the various products displayed, and the mechanical and scientific contributions to a doctor's armamentarium are examples of the ingenuity of the manufacturers. Cordial invitations are extended to visit these exhibits.

The most valuable vacation period for the summer will be that at Worcester June 4, 5, and 6.

Do not miss the Annual Dinner, for the social features are important adjuncts to scientific meetings. Your President will be especially pleased to see you there, for thereby you may show your appreciation of his devotion to your interests. He has secured speakers who will entertain you.

The general and local committees have worked diligently to provide for your entertainment.

Read the program carefully a second time and mark those features which you find therein of interest.

NEW ENGLAND SURGICAL SOCIETY

COMPRESSION FRACTURES OF VERTEBRAL BODIES*

BY JOHN P. BOWLER, M.D.,† AND JOHN F. GILE, M.D.†

COMPRESSION fractures of vertebral bodies are not an uncommon result of accidents in the fields of both recreation and industry. The work of Osgood and Rogers here in Boston, preceded by that of Brackett, Mixter and Wilson, and Sever, as well as by Cotton's earlier report of a case, should be sufficient reason for not bringing this discussion to the particular location of this meeting. But, based on our own experience, we believe that the practical importance of a discussion of compression fractures still lies in the frequency of failure of their early recognition, particularly when so commonly unaccompanied by signs of cord involvement. Complicating as they may an apparently trivial and not seriously disabling injury, the early symptoms may be ascribed to "back sprain", "muscle pull," "lumbago", and excite in either the patient or physician or both, no indication or perhaps justification for complete roentgenologic investigation, without which the diagnosis cannot be made. The delayed recognition forced by the continued or progressive development of symptoms results in diagnosis at a time when later pathological changes have rendered an originally simple problem less amenable to treatment.

This introduction well describes our own experience. Not many years ago we experienced the discomfiture of having a late diagnosis of compressed fracture established on a case dismissed by us as back sprain. In 1932 we treated nine cases of compressed fracture, which raised the question of the increasing prevalence of the condition and caused a review of our hospital cases dismissed with diagnosis of back sprain. Of twenty-two such records, nineteen included sufficient x-ray data to indicate a correct classification so far as exclusion of fracture was concerned. In three cases, however, there was definite history of injury of a type and sufficiency to have caused fracture, the disability could have accompanied fracture, x-rays were not taken, and strapping of the back constituted treatment. The fact that these three cases have not returned to us may or may not support the hope that the diagnosis was correct.

The mechanism involved in compression fractures is determined by the structure and motions of the spinal column which functions as two segments: the superior long flexible portion supporting the head and carrying the thorax and abdomen, and the inferior or short rigid portion carrying the lower extremities. The spinal column is comprised of superimposed vertebrae bound together by strong ligaments and held in alignment by strong active musculature. It is important to recall here that the posterior or lamina ligaments, as their name, ligamenta flava, implies, are yellow, elastic tissue which gives more and more under tension, does not tend to rupture—that they are not fibrous check ligaments as in the normal joint anatomy. The vertebrae articulate anteriorly through their bodies by the interposition of fibrocartilaginous disks and posterolaterally through articular processes. Movements exceeding the wide normal range unduly compress the vertebral disks or cause fracture of related bodies. Each vertebra consists of an anterior mass of spongy bone, the body, and a strong posterior portion, the neural arch.

The normal curvatures of the spine, responsible for the extreme elasticity of the column, result from the shape of the vertebral bodies as well as that of the intervertebral disks, both of which are thicker on the convex side of a curvature, and best developed in those regions of greatest flexibility of movement. In general, the greatest mobility occurs where normal flexion is the greatest, as in the cervical and lumbar regions. In the lumbar segment, due to the relative fixation both above and below and the immense leverage of the trunk working against the pelvis, occurs the great majority of compression fractures as a result of violent, exaggerated flexion. Such trauma is spent on the large cancellous vertebral body, resulting in a degree of compression commensurate with the force and the absence of subluxation of dislocation as a means of escape. Complete escape, meaning dislocation without fracture, is extremely rare; one interesting instance, followed by complete reduction, we shall show. The compression vice is maintained by the resistance of the strong neural arch and ligaments posteriorly and the common anterior ligament anteriorly.

Fractures of the spine are placed in two definite groups by the presence or absence of cord involvement resulting from the presence and extent of dislocation accompanying the fracture. Of forty-four cases of fractured spine treated by us in the past ten years thirty-six have been of the compression type involving one or more vertebral bodies. Of these thirty-six cases twenty-nine were of a simple compression type without sufficient dislocation or damaging complications to produce cord signs. In 1932, encoun-

*Read at the Annual Meeting of the New England Surgical Society, September 30, 1933, at Boston.

†Bowler, John P. — Surgeon, Hitchcock Hospital, Hanover, New Hampshire. Gile, John F. — Surgeon, Hitchcock Hospital, Hanover, New Hampshire. For records and addresses of authors see "This Week's Issue," page 1083.

tering nine such cases, we were impressed with the correctness of the general belief that such fractures were rapidly increasing, because of both the increasing prevalence of automobile accidents and the improved x-ray technique and facilities as offering less possibility of incorrect diagnosis. Our yearly incidence, however, does not indicate any unusual variation within the period under discussion. The incidence of these cases in our records ranges from three to six cases each year, increasing to six in 1926 and to nine in 1932. Of the ten-year total of forty-four, twenty-five cases have occurred in the last five years.

The seven cases complicated by dislocation are the only cases in which there was cord involvement. This varied from paresthesias of one lower extremity in one case to complete paraplegia in six cases with varying degrees of recovery, and with two deaths. Probably of no particular significance, these two deaths to which our fatalities were confined, followed fracture dislocation of the eleventh dorsal vertebra, in one of which autopsy showed death from pulmonary embolus one week after injury, and in the other puncture of the hemiazygos vein by the fractured vertebra and rupture of the liver. In these seven cases, fracture was confined to a single vertebra in four; multiple vertebrae were involved in three. Not included in our list of spinal fractures but of interest to report in connection with the mechanics of spinal injuries is one case of complete dislocation of the spine without fracture. Herzikoff of Los Angeles in discussing such a case states that after a careful review of the literature he found no other case reported. We should like to report a complete anterior dislocation of the spine without fracture occurring at the twelfth dorsal vertebra, with overriding downward over the bodies of the first and second lumbar vertebrae. The patient was a man of forty-eight years of age, injured in an automobile accident. X-rays taken one year after injury show a complete reduction. The patient is now living, five years after injury, with a complete paraplegia.

We have mentioned these cases associated with dislocation as a result of a more extensive action of the force causing the spinal injuries. This reduces our report to twenty-nine cases of uncomplicated compression fractures of the vertebral body with no cord involvement and with which associated injuries are rare. In the case of the patient in shock or with paraplegia there is not much likelihood of an atmosphere of trivial injury and casual management.

Automobile accidents have been credited as the origin of the increasing prevalence of compression fractures. A list of injuries involving violent hyperflexion is, in our experience, led by falls from trees, staging, etc., resulting either in the individual's landing feet first in a position of flexion of the trunk or falling across a raised object. Of our thirty-six cases eighteen, or just one half, resulted from such falls; eight, or less than one fourth, from automobile or train accidents; four were caused by a blow on the back with heavy pressure and occurring in logging accidents; three from tobogganing; and three of miscellaneous origin. Those resulting from tobogganing could easily be overlooked. In passing, the case mentioned earlier as not diagnosed by us occurred from this very cause. There was no accident, collision, or "spill". The compression resulted from hyperflexion on the rebound from a not-unusual bump with the patient sitting on the toboggan with thighs flexed and knees extended, an ideal set-up for such an injury if once considered. Oddly enough, we have yet to encounter our first case resulting from skiing—but not strange when one recalls the mechanics of the injuring force and the fact that flexion is the position of an efficient skier and that most falls are backwards or sideways in a position of spinal extension.

The location and extent of the fracture in these thirty-six cases are of considerable interest from the standpoint of the mechanics of the injury and because of the relationship of multiple fractures to the site. Of the thirty-six cases twenty-seven involved but one vertebral body with the inclusion of each vertebra from the eleventh dorsal to the fifth lumbar, inclusive, and with the involvement of the eighth dorsal in one instance. Of these twenty-seven cases twenty-one or seventy-seven per cent occurred in the lumbar spine, the second lumbar leading with fracture in seven cases. Six single fractures of one body were confined to the dorsal region, the body of the eleventh leading with four. In the cases of multiple fractures, of which there were nine, the extent was from the fifth dorsal to the fifth lumbar. In those cases there was involvement of two to four bodies. In all but four the fractures concerned contiguous vertebrae, the escaping vertebrae between the two segments of fracture varying from two to six in number. The above table gives

LOCATION OF COMPRESSION FRACTURES

Single

Vertebra	No. Cases	%
VIII D	1	
XI D	4	22
XII D	1	
I L	3	
II L	7	
III L	2	78
IV L	6	
V L	3	

Dislocation with cord involvement (3)—11%

Multiple

Vertebrae	No. Cases	%
Dorsal	5	55
Lumbar	3	33
Dorsolumbar	1	12

Dislocation with cord involvement (4)—45%

the incidence of fracture as occurring in the various bodies.

The age and sex incidence of these patients is of no significance, occurring with the same incidence as in general traumatic cases; twenty-five per cent were in women, and the ages of the entire group varied from fifteen to eighty-one.

To return to our twenty-nine cases of simple compression fracture without cord involvement, the diagnostic symptoms are not definitely pathognomonic of fracture. The constant symptoms are tenderness in the back, usually well localized over the spinous processes of involved vertebrae, and pain which usually follows immediately after injury and is continuous until a corrective position is maintained. The pain is located in the general region of the tenderness and may be girdle-like in character. There may be swelling at either side of the site of fracture. Kyphosis in the early and uncomplicated case is not present. The diagnosis is possible only by x-ray and the important issue in arriving at the diagnosis is a history of the manner of injury, a recollection of the mechanics of its production, a definite insistence with the patient of the possibility of the condition without further symptoms, and an insistence on complete roentgenological investigation.

The interval after injury which has occurred before some cases have been seen is significant of the casualness with which both physician and patient may regard a lame back following injury. Nineteen of our cases were seen within twenty-four hours. In thirteen the time averaged two and one half weeks; and in the four remaining, this interval was three months, seven months, eighteen months, and four years. These late cases had been treated with a short period of rest in bed, strapping, and finally came in with a complaint of persisting backache. The position of comfort in these injuries is moderate flexion and absence of lordosis; and is to be kept in mind in suspecting the diagnosis and in considering the transportation of such a patient.

The treatment obviously reverses the mechanics of the injury, and consists of hyperextension, for which many devices, tables, and maneuvers have been utilized. As a result of the muscle spasm which is present in the early cases immediate extension involves considerable traction on the abdominal muscles resulting in pain and extreme discomfort, often followed by nausea, vomiting, and, at times, by distention which may become serious. If extension is accomplished in a gradual manner, much of this discomfort can be avoided. The placing of sandbags and built-up slats under the patient has been one practical method for the institution of this extension. Except in the case seen immediately after injury we have found the early application of a cast to be often unsuccessful, usually producing such a degree of discomfort as to force its discontinuance. We have found the simplest method of producing gradual extension to consist of reversing the patient, head for foot, on a standard Gatch bed and using the knee elevator as a pressure producing extension of the spine; this mechanism lends itself very well to gradual application. We have extended the use of this method to a period of from ten to fourteen days, at which time all spasm has disappeared. The patient is then put on the ordinary Bradford frame in a canvas hammock and a body cast is applied with the spine in satisfactory hyperextension, taking care that padding in the region of the injury is sufficient to prevent undesired pressure over the area of fracture and to maintain extension later when the cast may be less well-fitting. Such casts are worn from ten to twelve weeks, with the patient ambulatory after the sixth or eighth week. On removal of the cast a Taylor brace is applied, which is worn for the balance of one year following injury, during the last six months of which the patient can usually return to ordinary work. We realize that there is a considerable difference of opinion as to the length of time that a patient should be immobilized with such an injury. In spite of some proponents of a much shorter period of immobilization, we feel safer on the program as stated. In the cases complicated by dislocation, with the early period of hyperextension has been combined leg or head traction or both as indicated. In general we have found the higher the fracture in the spinal column the more difficult the satisfactory reduction.

It would seem that no discussion of this type of fracture would be complete without reference to Kuemmell's disease. A considerable amount of literature on this entity has accumulated since Kuemmell's first article in 1891. It must be borne in mind that Kuemmell's articles preceded the practical use of the x-ray. The following sequence of events was described as necessary to make such a diagnosis:

1. Injury to the spine.
2. Period of disability.
3. Latent period.
4. Recurrence of pain and disability due to crushing of a vertebral body.
5. Arrest of the process by proper treatment.

In other words, to establish a diagnosis of Kuemmell's disease, an x-ray must be taken immediately after injury and found negative, and the patient later develops the subsequent course of events. Balin believes there is a microscopic dissolution of the continuity of the bone, which, without treatment, degenerates, absorbs, and collapses. Jones of Liverpool believes the fracture is reduced by manipulation of moving and therefore does not show, but without proper treatment crushes under the body weight and

the ensuing events occur. There seems to be but little doubt that so-called Kuemmell's disease is the late result of an unrecognized and therefore untreated crushing of the vertebral body.

CONCLUSION

1. Our cases of compression fracture of vertebral bodies without dislocation have been accompanied by no signs of cord involvement.
2. Such fractures resulting as they may from an apparently trivial injury may escape early recognition.
3. Kuemmell's disease is the late result of an unrecognized and therefore untreated case of such fracture.

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DISCUSSION

DR. EZRA A. JONES, Manchester, N. H.: *Mr. President and Gentlemen* — There are two points that are brought out in this paper which are extremely interesting to me. One is the failure to make a diagnosis in cases of compression fractures without cord involvement, and the other is the recovery of these patients without any fixation operation.

I think the failure to make a diagnosis is due to two factors. First, the failure to get a definite history as to how the accident occurred, and the second is the inadequate x-ray examination. Compression fractures always occur with the spine in flexion, and if it is found that flexion of the spine could have occurred during the accident, we should be suspicious of a compression fracture. Slight compression fractures can seldom be diagnosed in an anteroposterior x-ray, and lateral views should always be taken.

The use of the Rogers' bed makes the treatment very simple, but if that cannot be had hyperextension can be made with a blanket roll placed under the point of fracture. I have been using for several years the Whitman frame which I believe has many advantages. It is inexpensive, mobile, and quite effective. It can be made by any plumber and consists of a heavy gas pipe, about six to eight inches longer than the patient and as wide as the patient's shoulders at either end. It is curved upward from the bed to any degree desired, and at the point where pressure is desired the pipes are placed to within two inches of each other for about four to five inches at the point where the fracture occurred. The effectiveness of this is that the pressure comes on the transverse processes and not on the spinous processes. While on this frame, x-ray may be taken and any desired amount of hyperextension may be secured. After four weeks in this position a plaster corset is applied and this is worn for about four months when a back brace is instituted. It is rare to find a case treated this way which does not go on to recovery.

DR. JAMES W. SEVER, Boston, Mass.: I am very much interested in this matter of compression fractures of the spine because a number of years ago when I was doing more industrial surgery than now, I picked up a great many which had been unrecognized and untreated. I was not the first nor the only one, but I missed many. My imagination wasn't sufficiently good at that time to visualize what should be done for them, and I was content to treat these cases with some support or perhaps do something along the lines they had been having, or give them more treatment than they needed or had had.

It remained for others with a better visualization of the problem to sense that hyperextension was the thing that was really needed, particularly in the acute cases and you, of course, are familiar with Dunlop's method of forcible correction, and later Davis's (in Erie) under an anesthetic, with immediate correction of the wedging of the body, and later the excellent work that William Rogers has done with his fracture frame, as it is called. Since we have been shown the path that we should take with those cases, the problem has not been a particularly difficult one, so far as correction of the deformity goes.

It is perfectly amazing to see how the accordion will open with hyperextension properly applied, and with restoration in many cases to complete symmetry and anatomical outlines of the injured body, particularly, of course, in the lumbar region.

The medical profession, so far as the pathology of the spine has gone, has made more advances in the last five or ten years than have ever been made so far as I know. In the old days of railroad spines, or contusions of the spine, before x-rays, people were content to go on perhaps with traumatic neurosis or lame back and nobody thought much about it. Perhaps Kuemmell's disease was more frequent than it is now, and I think most of Kuemmell's works were written before the advent of the x-ray, so perhaps he couldn't tell that there had been a primary injury to a body of the vertebra.

The early treatment as we followed it, and as I think Dr. Gile has said, should be hyperextension, but hyperextension immediately applied is peculiarly uncomfortable and distressing and oftentimes leads to rather grave symptoms. Many of these people have a good deal of abdominal distention which is disturbing because you don't know whether they have an intestinal, or a bladder rupture, and in those cases they will not stand a hyperextension at all. Even after that condition has quieted down, forcible hyperextension applied may bring back symptoms which are extremely uncomfortable and painful.

In the old days when a person had a fractured back, everybody knew it, and when the diagnosis was made of fractured spine, a person was immediately, permanently incapacitated. The injury was associated with a section of the cord, dislocation of the cord, and paraplegia, and with symptoms which persisted because of the cord injury. The term "fractured back" today, except in the terms of psychology, is not particularly a serious condition. With the x-ray you can see compression fractures of the vertebrae or those fractures which Dr. Gile has shown which should not be called compression fractures so much as sprain fractures where corners are pulled off and where there is no frank compression of the body itself.

Today a fractured back simply means that there is a fractured lamina, or a pedicle, or a transverse process, or a sprain fracture of a corner. Even a fracture of the vertebral body doesn't mean prolonged illness or disability, and the reason is not only because of the x-ray studies but also of the sense with which these problems have been met in terms of treatment.

There is one other thing which I should like to speak of, and that is in the older people, who have marked hypertrophic arthritis, and who have injuries to the back from falls, from stagings, from roofs, from wagons, or whatnot. I have been unfortunate enough to make a diagnosis in several of these cases where there has been severe accompanying hypertrophic arthritis of the spine in compression fracture—there are severe distortions and asymmetrical conditions of the vertebral bodies which, on top of a severe injury to the back, are mistaken even by the x-ray man, as a compression fracture, and on review of the case the diagnosis has to be changed to "aggravated by hypertrophic arthritis," as the Compensation Commissioners like to say.

I have here something devised by one of the orderlies at the Boston City Hospital. It is an arrangement of a flexible steel plate. It is put under the patient and there is a ratchet arrangement here, that can be gradually turned up. I think it is known as the "City Hospital Hyperextension frame". The felt pad can be removed or padded, as you choose. Every day the plate can be turned up a bit, increasing hyperextension which is a better way than to do an immediate forcible correction. The apex of this plate should be under the deformity, and it can be readily and easily adjusted without discomfort to the patient. With this apparatus we can have hyperextension without danger of abdominal disturbance, to the point where the x-ray shows an excellent restoration of symmetry of the compressed vertebrae.

Dr. Robert B. Osgood, Boston, Mass.: I think that this is the most refreshing paper on compression fractures of the spine to which I have ever listened. It has an enormous amount of common sense in it and practical, simple methods of dealing with such lesions. It emphasizes the one important thing in relation to accidents to the spine, namely, that suspicion is the key that opens the door to diagnosis. We must think of this possibility always in injuries of the back, especially when we do not know the exact mechanism of the accident. This is very frequently the case.

It is refreshing particularly because not one word has been said about fusion operations for compression fractures of the spine and I think it should rarely be said, because it is obviously not necessary in the cases dealt with properly soon after the injury.

I think Dr. Sever is wrong. Dr. Davis gave the first clear exhibition of what could be done by manipulation. Dr. Dunlop and many other people have advised hyperextension. Dr. Davis deserves the credit of first demonstrating the possibility of opening up, as Dr. Sever so well called it, the accordion.

There is one thing I should like to bring out very definitely in this connection and this is that in the older cases it is very common after the diagnosis has been made, to advise a fusion operation because of disability and pain. If you study these cases carefully, you will find that a certain proportion of them have a perfectly good internal fixation by nature (drawing on the board) something like 33 per cent as I remember it of a large number of cases reported in St. Louis. The fusion operation would not then need to be done for the sake of fixing this region, because it is already fixed, and yet these cases often have pain and discomfort. The reason they have discomfort, as Wallace has well shown, is the fact of the postural defect in the spine caused by the wedge-shaped vertebra, and the discomfort does not come from the fracture, but from the disturbed posture.

You may in the older cases, without doing a fusion operation, certainly always before you do a fusion (as you may be obliged to do in working-men) produce by hyperextension compensatory curves above and below the seat of the fracture in the spine, which will reduce the postural defect and relieve the pain without any operative procedure.

I happen to have had to deal with a great many of the older cases where the lesion has been unrecognized and a correction of the postural defect by means of compensatory curves above and below has entirely relieved all symptoms without a fusion operation.

I think Dr. Gile and Dr. Bowler are to be warmly commended for the whole attitude of the paper and for the points that they have so well brought out.

Dr. James W. Sever: May I ask Dr. Gile a question? I should like to ask in relation to the borderline cases in compression fractures where there has been a frank compression fracture of the body and where there are at first and oftentimes persisting for some time, certain neurological symptoms in the legs, whether it is advisable in those cases, early or late, to do a laminectomy.

Dr. John F. Gile: I said nothing about the fusion operation because I don't know anything about it in the first place, never having done one and we haven't found the necessity for doing it. By correcting, putting on a proper brace or a cast, even in the old cases you get relief, so I have found no occasion for using it.

The suspicion of a flexion accident was brought out by Dr. Osgood in an article some years ago and is more or less the basis of our repeating it here. The suspicion of it is the thing to keep in mind. Suspect that there was a flexion and you have an idea of what may have happened.

Since this paper was written, a patient last week came to the x-ray man with a diagnosis from a doctor of some internal disease around the cecum and wanted a barium enema. Dr. Sycamore, with the story of the man's having had a fall last March, took an x-ray and found nearly complete compression fracture of the vertebrae.

We have not done laminectomies in these cases. With complete paraplegia we have felt that we would do no good—perhaps more radical treatment is indicated. The paraplegias and slight neurological symptoms have cleared up with traction.

THE NECESSARY REQUIREMENTS IN A MINIMUM DIET FOR INFANTS AND CHILDREN

BY JOHN LOVETT MORSE, M.D.*

AT this time when so many children are underfed and undernourished and when it is also so important that they be properly fed at as low a cost as possible, it seems worth while to review briefly what their nutritive needs are and how they can be met.

The minimum diet must contain proteins, fats, carbohydrates, salts and vitamins, and must also provide the requisite number of calories. Its bulk must also be taken into consideration. Attention must likewise be paid to its palatability and digestibility, points which are unfortunately frequently overlooked or forgotten. The relative importance of the caloric value of foods was undoubtedly overestimated in the past. Absolutely, however, it is as important as it ever was. The importance of the vitamins is now being relatively very much overemphasized, although, of course, no diet is complete which does not contain a sufficient amount of them. They are really no more important and necessary than certain amino-acids or even common salt, which are seldom mentioned.

It is stated in the reports of The White House Conference that "the correct procedure in constructing a diet must consist in first attempting to provide a complete assortment of the substances which sustain the chemical structure of the body and then adding combustible material in such amounts as will provide the necessary energy." This statement is undoubtedly true. Nevertheless, I am going to follow the custom, usual in the past, of taking up the caloric needs first, and considering the other needs later.

CALORIC NEEDS. These, in brief, consist of (1) The basal metabolic needs, which gradually diminish with age and, measured in body weight, are less in the fat than in the thin. (2) The needs for growth, which are greatest in infancy and in the prepubertal period. (3) The needs for muscular activity, which must necessarily vary enormously. (4) The needs for waste, which are relatively unimportant. It is evident how difficult it is to estimate these needs; even to get average figures. It is far more difficult to estimate them for the individual.

Most tables merely approximate what their makers think children ought to take and are consequently not of great value. Those which are based on what well, growing children actually eat are probably more nearly correct. Figures based on weight are probably less accurate than those based on height. According to the findings of The White House Conference, the caloric needs of boys gradually increase from thirty-five calories per inch at two years to fifty-five at seventeen years, while the needs of girls vary from thirty-nine to forty from six to fifteen years and between thirty-five and thirty-seven before and after this time. Approximate average figures as to the caloric needs of infants and children, based on those given by The White House Conference are as follows:

Infants. Sex has no influence.

1 month	500 Calories
6 months	800 "
1 year	1000 "
2 years	1200 "

Children	Boys	Girls
2-3 years	1200	1225
4-5 "	1525	1450
9-10 "	2250	2000
11-12 "	2325	2175
13-14 "	2625	2400
14-15 "	2900	2375

The figures given by Holt and Fales, modified by my own experience, are, I think, more nearly correct for children of twelve years or over. These are, for boys of twelve years, 2750 C, and for girls of the same age, 2950 C; at fifteen years, boys, 3750, and girls, 3700.

Finally, it must always be remembered, as stated in the proceedings of The White House Conference, that "each individual child has his own caloric needs which may vary from that of others of the same age and size. This optimum is such that the child grows properly, feels well, is mentally and physically alert and adjusts himself to his environment. Standards of food intake are of value in improving the welfare of children, but should not be used to the exclusion of the consideration of the problem of the individual child."

PROTEINS. As is well known, protein is the only one of the food elements from which old tissues can be replaced and new tissues built. No diet is adequate which does not contain enough protein to do this. Furthermore, the supply must be continuous, because proteins—i.e., amino-acids—are not retained in excess of the immediate requirements. The proteins are changed into the amino-acids before they are absorbed. Most of these amino-acids are interchangeable, but at least four of them are not. These are tryptophane, lysine, cystine and histidine. They cannot be synthesized in the body but must be taken in the food. Fortunately there is very little chance of an insufficiency of any of them, unless barely possibly of cystine, if a baby is on the breast or a baby or child is drinking milk. Practically, it is not necessary to consider them. Incidentally, on the whole,

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animal foods provide a better selection of amino-acids than do vegetable.

The storage of protein for growth will be defective on diets which do not contain sufficient calories, as some of the protein will be burned for energy. Proteins may yield glucose, fatty acids and fat. As much as fifty per cent may be converted to glucose. Protein is a wasteful source of energy. This is shown by a comparison of the specific dynamic action, i.e., the "cost of digestion", of the various food elements, which is 2.5 per cent of the fuel value of fat, five per cent of that of the carbohydrates, and ten per cent of that of the proteins.

The minimum protein need is the minimum amount of protein required to replace used tissues, build new, and pay the cost of digestion, assuming that the diet is otherwise sufficient, so that protein does not have to be used as fuel. The figures as to the minimum protein needs are probably more accurate for infants than for children. The breast-fed baby gets from 1.8 grams to 2.3 grams of protein per kilogram of body weight daily. This is equal to from seven to nine per cent of his total caloric intake. Various studies seem to show, however, that 1.5 grams per kilogram are enough. Babies taking the modern whole milk mixtures get from 2.6 grams to 5.8 grams of protein per kilogram, or from twelve to twenty per cent of their total caloric intake. This is probably too much, as has already been explained.

The figures as to the protein needs of children are conflicting and probably not very reliable. Holt and Fales found that from six years on, healthy, growing children took about 2.6 grams per kilogram. Various figures, which I have analyzed, seem to show protein needs of 3.5 grams per kilogram at four years, 2.5 grams at eight years, and two grams at twelve years. These amounts are probably larger than necessary. Other estimates are that from one to five years ten to fifteen per cent of the total calories should be provided by proteins, and in older children, five to ten per cent.

The chief symptom of protein insufficiency is failure to grow properly. If there is a marked insufficiency, nutritional edema may develop. In such cases, however, it is often very difficult to know how much of the edema is due to the protein insufficiency and how much to an excess of the other food elements and to a deficiency of vitamins, both of which are usually associated with an insufficiency of proteins. There are no pathognomonic symptoms of an excess of proteins. It is intriguing to think that an excess may injure the liver and kidneys, but, so far as I know, there is no proof that it does. Almost all that can be said is that proteins are an uneconomical source of energy, and that, therefore, it is wasteful to use them for this purpose.

Carbohydrates. The carbohydrates comprise the sugars and starches. Both must be broken down into monosaccharides before they can be absorbed. It must not be forgotten in this connection that raw starch is digested with much difficulty because of the indigestibility of the cellulose which surrounds the starch granules. Starch which is not broken down cannot, of course, be absorbed and, therefore, cannot be utilized. All the carbohydrates serve the same purpose in nutrition. This is mainly as a source of energy. Nevertheless, a certain amount of carbohydrate is necessary for the maintenance of life and to prevent ketosis. This minimal need is three grams per kilogram of body weight for infants and from eight to ten grams for children. The amount of carbohydrates needed to supply energy depends somewhat on the amount of fat in the food, which also serves mainly as a source of energy. Within reasonable limits they can be used indiscriminately for this purpose. In a general way, everyone agrees that the optimal amount of carbohydrates for the infant is from ten to fourteen grams per kilogram of body weight and from eight to ten grams for children. This means from forty to sixty per cent of the total caloric intake.

Carbohydrate insufficiency never occurs except in starvation. An excess of carbohydrate in the diet is, however, probably the most common mistake made in feeding children. The usual symptoms are indigestion and loss of appetite; if there is a great excess, glycosuria. There are no characteristic symptoms of an excess of carbohydrates. Those which are often attributed to it are really due to an insufficiency of the other food elements, especially of protein, and a deficiency of salts and vitamins.

Fats. The fats serve mainly as a source of energy. Some form of fat, however, enters into the composition of every cell of the body. A certain amount is, therefore, necessary for the maintenance of life and the building of tissue. There are no figures as to what this amount is. Fat is stored. If necessary, it can be synthesized from carbohydrate and protein. The caloric value of fat, by weight, is the highest of the food elements—9.3 vs. 4.1 calories per gram. It has the lowest "cost of digestion". The normal breast-fed baby gets from four to six grams of fat per kilogram of body weight daily, or fifty per cent of its total calories. A baby on the modern whole milk mixtures gets only fifteen to thirty per cent of its total calories as fat. This seems to me to be too little. Children on an ordinary diet take from three and one half to four grams of fat per kilogram of body weight, or about thirty-five per cent of their total calories. No one knows whether this is optimal or not. Much more than this is probably not fully utilizable and quite probably interferes with the utilization of calcium. Many of the fats are also

important because they are carriers of vitamins A and D.

The symptoms of fat insufficiency are not very definite. In the main, they are simply those due to an insufficient caloric intake. Except in starvation it is very improbable that not enough fat would be taken to carry on the vital processes. The symptoms which are usually attributed to an insufficiency of fat are really due to an insufficiency of the vitamins which are carried by the fats, namely, vitamins A and D.

Finally, to sum up, the optimal diet for children appears to be one in which protein makes up from ten to fifteen per cent of the total caloric intake, carbohydrate from forty to sixty per cent and fat from thirty to thirty-five per cent. The cheapest diet which will meet the nutritive needs is one in which ten per cent of the total caloric intake is provided by protein, sixty per cent by carbohydrate, and thirty per cent by fat.

VITAMINS. These accessory food factors are unquestionably necessary for the maintenance of health and normal growth. Their absolute importance cannot be overemphasized. Their relative importance is, however, being stressed altogether too much, largely because of the marvelous advertising and high-pressure salesmanship of the manufacturers of foods and drugs, who have taken advantage of one of the few opportunities to make good money in these hard times. We physicians have only ourselves to blame for being exploited. The general public cannot be blamed, because they cannot be expected to understand what is going on. The mistake which is being made is in assuming that unless a great effort is made to give the vitamins, babies and children will get an insufficient amount. Another mistake which is being made is in attributing many symptoms to avitaminosis which are in reality due to something else. Children still have sore eyes from other causes than a lack of vitamin A; loss of appetite may be due to indigestion as well as to an insufficiency of B. In general, in spite of all that has been written to the contrary, and although I know that I shall be reviled for saying so, I still think that the average child in the average American family runs very little chance of dodging a sufficient supply of the vitamins. The case is, of course, very different among the very poor and among those who eat a very one-sided diet. Babies on the present low fat mixtures may also perhaps get an insufficient supply of A and D, especially if they are kept indoors. Furthermore, with the present-day methods of feeding, all babies that are not breast-fed are very likely to get an insufficient amount of C. It is very fortunate that an excess of the vitamins does no harm. If it did, many of our babies and children would certainly be in a parlous state. The manifestations of a marked insufficiency of the various vitamins are now pretty well known. What is needed is a better knowledge of the symptoms of a slight insufficiency.

Vitamin A. An insufficiency of this vitamin causes a specific change in the epithelial tissues. Stratified epithelium is substituted for the normal in various parts of the body, notably in the respiratory, alimentary and genito-urinary tracts. It is argued that this change results in an increased susceptibility to infection. Theoretically, it should. The best controlled clinical studies, however, seem to show that it does not. It would seem as if it ought to cause some disturbance of normal function. Certain changes in the skin have been described in infancy. A marked insufficiency causes night blindness and xerophthalmia. Vitamin A is stored. The limit of the capacity to store is not easily reached.

The common sources of vitamin A are milk, butter, eggs, animal and fish oils, and the green vegetables. Cereals and muscle tissues contain but little. Lard, vegetable fats and oils do not contain it.

Vitamin B. This vitamin is made up of a number of different factors. The two most important ones are known as B-1 and B-2, or G. B-1 is the antineuritic and G the antipellagric or antidermatitic. The whole subject of the subdivisions of B is most confusing.

A marked deficiency of B-1 causes beriberi. A moderate insufficiency is said to cause anorexia, hypertonicity, and impairment of growth. There is probably nothing specific about the impairment of growth. This is presumably simply the result of the diminished intake of food because of the loss of appetite. Much has been said recently about loss of appetite in infancy and childhood as a symptom of an insufficiency of vitamin B in the diet. I am inclined to think that in the vast majority of instances the loss of appetite is not due to an insufficiency of vitamin B but to some unrecognized disease, improper hygienic surroundings or care, or some disturbance of the digestion from some other defect in the diet. At any rate, I have never been able to restore the appetite by increasing the amount of vitamin B in the diet.

The principal sources of B-1 are whole grains, wheat germ, yeast, the glandular organs, and the yolk of egg. Other sources are tomatoes, raw cabbage, fresh spinach and the legumes. Milk contains relatively little.

Vitamin G. (B-2) A marked deficiency of G causes pellagra. A moderate deficiency results in various forms of dermatitis, and is said to cause loss of appetite and impairment of growth. What has been said as to loss of appetite and impairment of growth in relation to B-1 applies also to G.

The sources of vitamin G in order of efficiency are liver, yeast, meat, milk solids, egg yolk, and whole grains.

Vitamin C—the antiscorbutic vitamin. A marked deficiency of this vitamin is, as is well known, the cause of scurvy. Symptoms of a moderate deficiency are defective development of the teeth, with dental caries, diminished resistance to infection, changes in the blood vessels and a tendency to hemorrhage, loss of appetite and anemia. It must be remembered in attributing defective development of the teeth and dental caries to an insufficiency of this vitamin that the teeth of guinea pigs grow continuously while those of man do not. Conclusions based on experiments on animals may not be, therefore, justified for man. It is very difficult to prove, moreover, that a deficiency of vitamin C really diminishes resistance to infection in man.

It is hardly necessary to discuss the sources of vitamin C. Every baby gets either orange juice or tomato juice nowadays, whether it needs it or not. Scurvy is, however, a medical curiosity in the breast-fed. It was very uncommon in babies fed on raw cows' milk, although, of course, it is well known that the amount of the antiscorbutic vitamin in milk varies with the time of year and the food of the cows. Now, when all babies are fed on pasteurized, boiled or dried milk, an antiscorbutic is a necessity.

Vitamin D—the antirachitic vitamin. So much has been written about this vitamin, rickets, fish oils, viosterol, irradiated foods and ultraviolet irradiation during the last few years that I, for one, am tired of it all. The medical journals are all cluttered up with advertisements of antirachitics and my mail is full of samples. Fortunately, I can usually keep the detail men out. There is no doubt, of course, that an insufficiency of vitamin D or of its activator, the ultraviolet rays, will result in the development of rickets. It undoubtedly also has something to do with the development of spasmophilia and with defective development and caries of the teeth. There is also no doubt that vitamin D will cure these conditions. It must be remembered, however, that in all these conditions a proper supply of available Ca and P is as necessary as the vitamin. It is also true that a slight amount of rickets is not a serious condition. Too much stress is being laid upon it. Furthermore, as Eliot and her co-workers have recently shown, the clinical diagnosis of mild rickets is often wrong.

There are many sources of this vitamin. Fish oils contain the most. Animal fats contain some, vegetable fats little or none. The amount in egg yolks varies. The grains contain none. Oatmeal is said to do harm. Cows' milk contains little. It, like other foods, can be irradiated. Vitamin D can also be introduced into milk by feeding irradiated yeast to the cows. The ergosterol in the skin can be activated by exposing the body to the sun's rays or to those from various lamps.

Fortunately there is practically no danger of getting an excess of vitamin D clinically, although experimentally serious results have been produced by an excess. It is lucky that this is so, because, if it were not, a good many babies that are taking various fish oils, viosterol, irradiated foods, and going about naked would be in serious difficulties.

In this part of the world it is probably advisable to see that babies get vitamin D in some form during all but the summer months. In fact, there is no doubt that, by and large, in spite of all that I have just said, babies have been much benefited by the general use of vitamin D during the last few years. There seems to be no valid reason, however, for giving it to children as a routine procedure.

To sum up: Practically, if a child gets a reasonable amount of milk, an occasional egg, some meat, and whole grains, it is very unlikely to lack vitamins. If the milk is not raw, a baby needs an antiscorbutic. It is a good thing for a child to have some vegetables as an additional safeguard. Spinach and other "leafy" vegetables are not, however, as necessary as many would lead us to believe. It is advisable to give babies vitamin D in some form during the winter months.

SALTS. It is impossible to discuss the salts in detail in a paper of this sort. The subject is altogether too complicated and has too many ramifications. The food must contain certain salts in sufficient amounts. Furthermore, these salts must be present in such a form that they can be absorbed and utilized. There may be plenty of them and yet they may be practically useless because of the form which they are in or because of the presence of or lack of other factors. For example, the absorption of Ca and P depends on the relative proportions of each and the reaction of the intestinal contents; the utilization of Ca depends on the amount of vitamin D and the activity of the parathyroids; and so on. Furthermore, the availability of the salts also depends on the various amounts of the various food elements—fats, carbohydrates and proteins—in the diet. In general, an *excess* of one of the salts is unimportant, unless it interferes with the absorption or utilization of some other salt. The excess is simply not absorbed or it is promptly eliminated. A *deficiency* of one or more of the salts is the important thing.

The salts which are of practical importance are those of Ca, P, Fe, and I. We can safely forget copper, cobalt and manganese! It is very difficult to state just what the needs for these elements are because of the complicating factors just mentioned. Roughly, a child needs one gram of Ca and one and half grams of P per day. It is hard to see how a child that is taking milk, eggs and cereal can fail to get enough.

A retention of one half a milligram of *iron* a day is required during the first six months, of one milligram during the rest of the first year, and of two milligrams during the rest of childhood. In general, it is still safe to assume that the store of iron in the liver of the average infant is sufficient to make up for the deficiency of iron in the milk during the first six months. Milk is, of course, notoriously low in iron. Eggs, prunes and green vegetables are the best source. The relative importance of the latter has, however, been much exaggerated. This has been shown by Schlutz and his co-workers in a recent paper.

The need for *iodine* is one tenth of a milligram per day. In this part of the country it does not need to be considered. This is especially true when we remember that almost every baby and child in this vicinity is taking syrup of hydriodic acid.

WATER. It is hardly necessary to take up the water needs in detail. It must be a very unusual occurrence for either a baby or a child not to get enough water to cover its metabolic needs. In many instances, however, it would be better for it to get more.

SUMMARY. I have endeavored to show what a child needs and must have in its diet in order to be well and to grow satisfactorily. It is evident, it seems to me, that the individual child needs just so much of the various food elements, salts and vitamins, no more, no less. If he takes more, it is wasted; if he takes less, he must suffer for the lack of it. The minimum diet is, therefore, the one diet which meets the child's needs, that is, the ideal or optimal diet. If he gets less than this, he will not starve to death and may not show any striking evidences of disease, but he cannot thrive and develop as he should. This minimum diet should be the aim and guide of those who are prescribing the diets for the poor and undernourished children. This diet may vary very much in cost according to the amount of knowledge and skill which are used in its selection and preparation. It need not be, and ought not to be, expensive. In fact, it may and should cost very much less than many diets which do not meet the nutritive needs nearly so well.

THE USE OF ERGOTAMINE TARTRATE IN MIGRAINE*

BY WILLIAM G. LENNOX, M.D.†

DRUGS used for the relief of migraine are in most part sedatives, burying the pain but not righting the condition responsible for the pain. Moreover, with many patients the headache seizures are so severe that even strong sedatives do not give relief. Any non-sedative drug which, in the majority of patients, will cut short a severe migraine attack deserves thorough clinical trial and careful experimental study.

Ergotamine tartrate (which is sold under the trade name of "Gynergen") was called to my attention by Dr. Stephen J. Maddock, who had used it with dramatic results in a patient with migraine whose attacks had not been relieved by any of many treatments tried. A search of the literature disclosed several communications on the subject from European authors. Maier[1] in 1926, in an article dealing with the effects of ergotamine tartrate on the sympathetic nervous system, says that he has used it successfully in cases of pruritus, migraine and epilepsy. Cornil[2] recites a case whose migraine headaches could be stopped by the oral administration of the drug. There are also case reports in theses which cannot be consulted, viz., Déclaifayt of Bordeaux in 1926, and Cantils of Montpelier in 1929.

The first and also the largest series of cases has been that of Tzanck[3, 4, 5]. In 1928, he reported 12 cases and in 1931, 101. In his first report, he states that of eight cases of typical and chronic migraine resistant to all previous treatment, all had been favorably influenced. Of three other patients with almost continuous headache, two had been almost completely cured, and one had discontinued treatment because of painful menstruation. One other patient with ophthalmic migraine had been relieved of headaches but not of the ocular symptoms. Tzanck's analysis of results in his last reported series of 101 cases is disappointingly brief and indefinite. He states that 15 out of the 44 members of the group which he calls "typical" had been "cured" and the condition of nearly all ameliorated, and that results were even more favorable in patients having a migraine state. By this term he means attacks which are less clear cut and violent. Some patients were apparently "cured"; some had attacks at longer intervals; some developed a tolerance for the drug, requiring larger and larger doses for relief. Tzanck gave the ergotamine by mouth, from one to as high as six mg. a day, or subcutaneously 0.5 mg. twice a day. Besides treating individual attacks, he speaks of the prophylactic use of daily doses.

Trautmann[6], at about the same time as Tzanck.

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reported the use of ergotamine tartrate in 30 cases of migraine. The results are not tabulated. The author merely states that, except in individual instances, one or two mg. of the drug by mouth would begin to relieve the headache in about one-half hour, and that it was without effect in headaches associated with organic brain injury. An indifferent agent was given first in order to rule out the factor of suggestion. Targhetta[7] reported successful termination of attacks in one case. Ignelzi[8] had favorable results in six cases. He gave the drug intramuscularly in 0.5 mg. doses. Kottmann[9] reported treatment in five patients. Subcutaneous medication relieved each attack. Little success attended oral or rectal administration. One patient, however, who had monthly headaches at the time of menstruation, took six mg. of ergotamine by mouth daily for five days before headache was expected, and for four months had no attack. Riley[10], in his monograph on migraine, states that Tilney recommends ergotamine tartrate, either alone or combined with bellafoline.

Mignot[11] has no cases to report but reviews the literature, naming the various medical conditions in which ergotamine has been used, and discussing the dosage.

These authors do not mention serious untoward results from the use of ergotamine tartrate. The drug has been used extensively by obstetricians and gynecologists to control uterine hemorrhage, and by internists in the treatment of exophthalmic goiter, glaucoma, hypertension, auricular tachycardia, and pruritus of hepatic or of renal origin. Zorn[12] detailed four cases (one his own and three from the literature) in which use of ergotamine tartrate was followed by symptoms suggestive of beginning gangrene of the extremities. Oginz[13] reported a case of puerperal sepsis in which six drams of fluid extract of ergot and 22 mg. of ergotamine were given in a period of fourteen days. The patient developed gangrene of the feet. Three patients with exophthalmic goiter in which cerebral vascular accidents occurred in the course of treatment were reported by Labbé[14] and his associates. In these various reports, it is not clear that the injections were mainly responsible. In addition to these grave sequelae, authors speak of nausea and vomiting and sometimes of precordial pain which follows administration of the drug.

Because of the favorable reports in the literature as well as the surprising results in my own preliminary trial of the drug, I have, in the past eighteen months, used ergotamine tartrate for all migraine patients who have been willing to experiment with it.* The experience is too short and the cases too few to make detailed analysis profitable. I shall give such general results and experiences as will aid those who wish to make trial of the drug.

To date, ergotamine tartrate has been given to 45 patients (6 men and 39 women) while they were suffering from a headache of the migraine type. Abrupt termination of the headache in question occurred in 40 of the 45 patients. Of the remaining patients, three had no relief whatsoever, two patients had no immediate relief but the attack was apparently shortened by two days.

In migraine, emotional factors often play a prominent part. Hence, an attitude of skepticism on the part of the clinician toward apparent immediate results is essential. Perhaps "suggestion" and not medicine stopped the pain. In order to minimize suggestion, patients were warned that they were to be given the ergotamine (which sometimes helped headaches) as an experiment. Most of them considered this just another straw to be grasped at. The uniformity of the time interval between injection and relief, the fact that this interval was different for intravenous and subcutaneous injections (which difference patients did not expect), the fact that in the past other drugs and procedures had not been even temporarily effective for most of these patients, and, finally, the absence of relief from the injection of water (tried in a few patients), argued against suggestion as the explanation for the relief.

Of course, a certain small proportion of patients will obtain relief in the first trial of any new medicine or procedure. For nearly 90 per cent of migraine patients to obtain abrupt release from pain with the initial use of a drug is outside my experience. To some of the patients, the dramatically abrupt and complete termination of the headache, the sudden release from a state of misery, seemed almost miraculous. In the experience of these patients, an attack once aborted, rarely returned. Unlike hypnotics, the relief persisted beyond the period of action of the drug. After the pain had disappeared, there was usually a feeling of lassitude. The following description of events from a patient's report is fairly typical.

"The headache began to make its appearance about four o'clock in the afternoon and gradually increased in severity until about seven o'clock when I was quite sure I was in for a 'regular' one and so decided to make use of 'Gynergen'. I had it injected (0.5 mg. subcutaneously) into my right arm and went to bed. A half an hour previous to this I had consumed a generous dinner as I was, as usual, very hungry. After being in bed for about fifteen minutes, I began to feel very nauseated and at regular intervals for about twenty minutes I vomited continuously. Being somewhat exhausted, I fell asleep and slept until about 3 A. M. On waking, to my great surprise and relief, the headache had completely vanished. I felt extremely tired, as if I had had a very active day out-of-doors, but it was a pleasant feeling of fatigue and I turned over and slept until about 7:30 A. M. I was able to

*The ergotamine tartrate ("Gynergen") used in these trials was supplied by the Sandoz Chemical Works of New York City. Seven of the patients were treated by a colleague; Dr. T. J. C. von Storch, Dr. Stephen Maddock, Dr. H. S. Forbes or Dr. Philip Solomon.

go to my Saturday morning class and to carry on my usual activities during the day which is the first time within the experience of these headaches that I have been able to do so."

In the majority of cases, nausea or vomiting, or both, occurred before the headache began to lessen. Vomiting was not, however, a necessary preliminary to relief. In these patients relief began after the administration of the drug in the following period of time; viz., intravenously, 15-30 minutes; subcutaneously, one to two hours; orally, two to three hours. The order of effectiveness was the same; intravenous or intramuscular, most; subcutaneous, next; and oral, a poor third. Although dosage by mouth was double that used by needle, patients whose headaches were severe, were not ordinarily helped by oral administration. A few found that tablets taken by mouth would stop the milder headaches or make severe ones more bearable.

Ergotamine tartrate is marketed in ampules containing either 0.25 mg. or 0.5 mg. of the drug, and in tablets containing 1.0 mg. The recommended single dose is 0.5 mg. subcutaneously or 1.0 mg. by mouth. Because patients vary in their reaction to the drug, it is probably wise to give but half the full subcutaneous dose at the first trial. If this is well borne but not effective, the full dose can then be given. The dosage can be repeated after an interval of two to three hours. For a prompt sustained effect, I prefer to give 0.5 cc. (0.25 mg.) intravenously and, at the same time, the same amount subcutaneously. The dose should be varied in order to find the least amount which is effective. It is presumably more effective if given early in the attack.

Besides the nausea and vomiting experienced by many of the patients, three complained of dyspnea and a sense of constriction in the chest. One of these patients, Mrs. T., who received 0.5 mg. intravenously, had, along with this, symptoms of angina, and an increase in blood pressure from a preinjection level of 142 systolic and 90 diastolic, to a height of 180 systolic and 160 diastolic. Although Meakins and Scriver[15] gave as high as 18 mg. by mouth daily for 20 days to patients with hypertension, it would seem wise to use ergotamine cautiously in patients with vascular disease. Following injection, there is usually increase in systolic and decrease in pulse pressure and bradycardia. After the headache ceased, a feeling of fatigue or muscle soreness was common. One patient believed that menstruation was made more profuse. Pregnancy is, of course, a contraindication.

What has been said applies to the treatment of headaches after they have begun. Can ergotamine be used to prevent the seizure? Unfortunately, the danger of ergotism makes the daily administration of ergotamine over a prolonged period questionable, and most patients do not have attacks with such regularity that treatment can be concentrated in a preheadache period. Several of the patients had headaches in spite of taking 3 mg. by mouth daily. One of these, by taking 9 mg. daily, apparently skipped two headaches, but finally had one in spite of this large dosage. I doubt the feasibility of preventing attacks by this medicine.

Although the immediate results in most patients are gratifying, the question of the value of continued use of ergotamine is important. Some of the European authors speak hopefully of a lessening, or disappearance of attacks. One of our patients who was having headaches at about weekly intervals, since receiving three injections, has not had an attack in two months. We doubt, however, if in our average case the interval between attacks has been altered.

Certain of our observations temper enthusiasm concerning long range prospects. Patients whose attacks are terminated when treatment is first instituted may later on fail, at times, to obtain relief, or else the interval between attacks may be shortened. There is some evidence that in epilepsy the use of antispasmodic drugs may act only as a dam, postponing but not, in the long run, preventing attacks. The same may be true in certain severe cases of migraine. At any rate, evaluation of the clinical worth of the drug must await a longer experience.

The experience of the eight patients whose headaches were frequent and incapacitating, who had had no relief from the usual drugs and who began to use ergotamine more than six months ago will be given briefly. The first six patients are women.

A housewife, aged 54, has had frequent incapacitating hemicrania with nausea and vomiting since childhood. Besides various medical treatments, she had submitted to removal of her gall bladder and of her cervical sympathetic nerves, without even temporary relief. Headaches which lasted two to three days recurred every week or ten days. In addition, in recent years she has had almost daily moderate headaches for which she uses headache powders. Ergotamine tartrate has been used subcutaneously for 18 months. Each time it stops the headache, but on some occasions the pain has returned, and there has been a tendency for attacks to recur more frequently. She now takes 0.5 mg. subcutaneously almost daily. Her suffering is greatly reduced, but she is still in the coils of this disease.

A high school Latin instructor, aged 36, with a history of migraine since childhood, has an attack about once a week which lasts for two days. Vomiting, which is profuse, was formerly followed by relief of headache but is not now. She has very poor posture and musculature, visceroptosis and constipation. Each of the several dozen attacks which have assailed her in the last nine months has disappeared about 75 minutes after subcutaneous injection of 0.3 mg. of ergotamine tartrate. Vomiting is violent. After the headache subsides, she is weak and does not regain her strength before the next attack occurs. The interval between headaches has not changed. The individual attacks have been robbed of their terror, but her general situa-

tion is, at present, no better than when treatment was started.

A postgraduate college student of 32 years has had incapacitating headaches lasting two to five days since adolescence, the frequency in recent years being once or twice a month. Each of the attacks treated in the last year was aborted, although she has not taken an injection for every headache because of the unpleasant gastric symptoms. In recent months, the interval between attacks has been greatly increased, possibly as a result of dietary treatment.

A bank official, aged 44, has had headaches at about monthly intervals for 11 years. With each attack she has been away from her office under morphine medication for from two to six days. In the past 15 months, each of her attacks has been terminated by an injection of ergotamine, so that each time she has lost no more than a day from work. Headaches are somewhat more frequent than in the pre-treatment period.

A housewife, aged 41, who conducts a beauty parlor, has had severe headaches since her marriage, 21 years ago. They come about three times a month, each lasting 24-48 hours. There are also other mild headaches, not migrainous in type, which come frequently but irregularly. She has had mild hypertension for years, and six years ago had an infarct of a kidney. She is the patient already mentioned, who has anginal symptoms after intravenous or subcutaneous injections. She has been under observation for a year. Although each injection gives abrupt relief, her cardiovascular symptoms have forced resort to oral administration. This method is not nearly so effective but 2 mg. shortens the period of incapacity. The patient's "ordinary" headaches are not helped by ergotamine. In the last six months, probably as a result of a vacation, she has had but few attacks.

A housewife and physician has had incapacitating headaches lasting two to three days since girlhood. A headache always accompanies menstruation and may appear in an interval also. She has taken ergotamine for nearly two years—except during the nine months of pregnancy when she was free of headaches. Each headache has been aborted. The interval between headaches is reduced to ten days or two weeks.

A male physician of 47 years for the last three years has had attacks of right hemicrania, coming two to five times a week, each attack lasting two to six hours. The diagnosis in this case (the only one of the series) is questionable. He does not have nausea or vomiting. There is localized tenderness of the scalp and the pain radiates into the right arm. Attacks were uniformly aborted for three months by ergotamine tartrate, taken either by mouth (the method preferred by the patient) or by injection. In the last three months, however, repeated trial has been without avail.

A male factory worker, aged 28, has had ophthalmic migraine (with an aura of homonymous hemianopsia) about once a month for nine years. The aura has, on seven occasions, ushered in a convulsion in place of a headache. The stellate ganglion of one side was removed by Dr. Donald Munro 18 months ago and the patient was free of headaches for several months. Ergotamine has been taken for many of the attacks in the last year with abortion of each attack. He has not had a severe headache with visual aura for four months. Milder attacks are controlled by ergotamine taken orally.

On the basis of this preliminary experience, it seems probable that patients with frequently recurring and very severe headaches may not find permanent help in ergotamine tartrate. Those mildly affected, who obtain relief from one of the sedatives, will not care to change. But many persons in a middle group, who have attacks of moderate severity and who do not get relief from the usual remedies, may find ergotamine tartrate a godsend. The distracted physician to migraine patients has a new weapon in his armamentarium. These remarks refer only to the medicine as a palliative, giving symptomatic relief for individual attacks. The prevention of attacks, the "cure" of migraine is a different matter. The successful use of a drug should never displace efforts to correct underlying physical or emotional abnormalities.

DISCUSSION

What is the mechanism by which, on occasion, ergotamine tartrate so decisively puts the pain of migraine to flight? Among other actions, the drug stimulates the contraction of smooth muscle and depresses the activity of the sympathetic nervous system. In the latter respect, it is the antagonist of adrenalin. The authors who have been quoted accept the theory of vascular spasm as the cause of migraine and assume that ergotamine paralyzes the motor sympathetics and thereby relieves the spasm and the pain. On the other hand, one might assume that small doses of ergotamine act on sensory nerves with elimination of painful stimuli but without necessarily altering the calibre of vessels and the cerebral blood flow. In this connection, the relief of pruritus by ergotamine tartrate is suggestive. The time interval between injection and relief is the same for both pruritus and headache. As yet unexplained is the fact that after subcutaneous injection, blood pressure changes and uterine contractions begin almost at once but relief of headache does not begin for nearly an hour.

In the case of one of our patients, we have therapeutic evidence against the exclusive importance of the sympathetic nervous system. She is the patient already mentioned, who had complete bilateral sympathectomy by an excellent neurosurgeon in Baltimore. The operation did not influence headaches, but ergotamine aborted each attack. With the sympathetic pathways obliterated, how did the ergotamine act?

Does ergotamine help only patients with migraine? This question of specificity is important for etiological studies. Trautmann[6] and Kottmann[9] state that ergotamine does not influence non-migrainous headaches. Indeed, the latter contends that if ergotamine is not effective the patient is vagotonic. In our experience, however, ergotamine may terminate headaches which cannot be classed as migraine.

Certainly the theory of the mechanism by which the drug works requires experimental testing. We are interested in ergotamine tartrate principally because of our hope that study will

disclose the physiological mechanism by which it affords such abrupt relief. If we can learn why it stops a headache, perhaps we may learn why the headache started, and thus have a basis for more rational treatment. A group of us are conducting a clinic for patients with migraine at the Boston City Hospital and carrying out a coöperative attack on the problem. The physiological effect of ergotamine tartrate on patients, especially as regards cerebral circulation, will be the subject of future communications.

CONCLUSIONS

Ergotamine tartrate has been given by intravenous or subcutaneous injection to 45 migraine patients while they were having a headache.

Abrupt termination of the initial attack treated occurred in 40 of the 45 patients. Eight of the patients had had frequent, incapacitating headaches for many years, had not found relief by other drugs or treatment and have used ergotamine tartrate for six months or longer. Seven of these have had almost uniform relief for individual attacks. Of these seven, four are having attacks at more frequent and three at less frequent intervals than before medication was started.

Study of the mechanism by which ergotamine tartrate so often gives such dramatic relief is in progress.

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THE PARENTERAL ADMINISTRATION OF PARALDEHYDE FOR THE CONTROL OF PAIN AND CONVULSIVE STATES

BY ALLEN S. JOHNSON, M.D.*

PARALDEHYDE was introduced by Cervello in 1882 but its use as a hypnotic has been greatly hampered by reason of its disagreeable odor and taste, which may be constantly apparent to the patient for twenty-four hours after administration. Rectal administration has only partly overcome this difficulty as the rapid and persistent elimination by the lungs gives rise to considerable discomfort. Because of these drawbacks the drug might have fallen into disuse were it not for the rapidity of its action and the large margin of safety between the hypnotic and the fatal dose. Five to 10 cc. diluted with water or some other vehicle represents the usual hypnotic dose by mouth or rectum. The lethal dose, on the other hand, is probably not less than 50 cc. though reports of fatal paraldehyde poisoning are difficult to find. Bastedo[1] states that death has occurred from 60 cc. given by rectum but also reports a case that recovered after the administration of 100 cc. Sollmann[2] says, "Its acute toxicity is low so that one hundred grams produced only very prolonged sleep." I have safely given 60 cc. intramuscularly in the course of twenty-four hours to relieve the pain of carcinoma of the bladder which could no longer be controlled by morphine. The safety of paraldehyde probably lies in the fact that it is rapidly eliminated and does not depress the circulatory and respiratory systems until very high concentrations have been reached. But the unpleasant characteristics of the drug have limited its use largely to asylum practice and to the control of delirium tremens where effectiveness and safety of dosage are more important than the aesthetic objections of the patient.

In 1912 Noel and Souttar[3] reported the intravenous use of equal parts of paraldehyde and ether in normal saline. "Deep anesthesia can be very rapidly produced,—hypnotic effect is excellent. It is well to give with an equal amount of ether. The action of the drug is very rapid, hence best given diluted. 5-15 cc. of paraldehyde is mixed with an equal quantity of ether; this mixture is dissolved in 150 cc. of cold solution of sodium chloride (1%) in sterile water. Inject the solution cold or at not exceeding 25 C. Fildes and McIntosh Injection apparatus is used. Five seconds after injection the patient tastes paraldehyde; in forty seconds unconsciousness occurs; and in ninety seconds

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the corneal reflex is absent and anesthesia complete. By this time 5 to 10 cc. of paraldehyde will have been given and some minor operations may be performed; but as the drug is rapidly excreted by the lungs usually the whole 15 cc. will be required. The method is practically safe and it has been used in cases of grave cardiac and pulmonary disease with perfect success."

Atkey[4] the next year described a similar technique for the administration of paraldehyde in tetanus. In this case 15 cc. each of paraldehyde and ether in 150 cc. normal saline were given daily for eight days. The patient recovered.

Bastedo[1] mentions the intravenous use of 2-5 per cent paraldehyde in normal saline as a general anesthetic for short operations. He remarks that "anesthesia ceases when the infusion is stopped but sleep lasts for several hours". In none of the other half dozen standard texts on pharmacology which were consulted could reference be found to the parenteral administration of paraldehyde. Examination of the *Index Medicus* and the *Quarterly Cumulative Index* for the past ten years failed to reveal any references to the parenteral use of paraldehyde for the control of pain and convulsive states. Though no claim of originality is made it seems worth while to report the following observations because of the usefulness of this drug in properly selected cases, especially where morphine has proved ineffective.

My first experience with intramuscular paraldehyde was in the case of a patient who had become maniacal after a herniorrhaphy. Morphine in the usual hypnotic doses was ineffective and physical restraint was deemed unwise because of the operative wound. Eight cc. of paraldehyde were injected deep into the buttock as rapidly as possible. The patient lost consciousness before the syringe had been emptied and a peaceful sleep of some six hours followed. Equally good results were obtained in two other cases who had become unmanageable. A case of acute alcoholism who had become maniacal was subdued by this method so that gastric lavage could be carried out.

Encouraged by these results, the drug was next tried in the terminal convulsive stages of vascular hypertension which could no longer be controlled with morphine. Complete relaxation was obtained at once and the patient fell into a quiet sleep for several hours. This procedure had to be repeated a few days later but each time the convulsions were satisfactorily controlled. By this method it has been possible to control the convulsions of several other cases of advanced vascular disease. Intramuscular paraldehyde has not, to my knowledge, been used in eclamptic convulsions but it would seem worth trying as its action is rapid and safe.

Because of its rapidity of action it was given to relieve the pain of a coronary thrombosis which large doses of morphine had failed to control. This patient lost consciousness before the needle was withdrawn from the buttock and a quiet sleep lasted for several hours. The cardiac pain on awakening was negligible in this case. This procedure has been used in five other instances of coronary thrombosis where morphine had failed to bring relief. Equally good results were obtained though the loss of consciousness did not result so quickly in all these cases as in the first one. However, the patients admitted relief of pain within two minutes of administration and were asleep within five minutes. So striking is the result with 8 to 10 cc. of paraldehyde that one almost hesitates to subject the patient to the extra twenty minutes of suffering while waiting to see if the morphine will be adequate.

There is one serious drawback to intramuscular paraldehyde in addition to the unpleasant odor imparted to the breath. It is highly irritating and its injection is accompanied by severe burning pain. If enough is given to produce immediate unconsciousness, however, very little suffering is caused though some soreness persists. When paraldehyde is injected intramuscularly the undiluted U.S.P. drug is used. Eight cc. may be considered an average dose for an adult weighing about 140 lbs. I have frequently given 10 and occasionally 12 cc. to very large or obstreperous patients. The objection to these larger doses lies not in their toxicity but in the discomfort and difficulty encountered in injecting such a large amount of fluid into the muscles, especially when that fluid is as irritating as paraldehyde. Even with these larger doses, however, I have found no evidence of necrosis. In fact, the soreness at the site of injection is surprisingly transient considering the comments occasioned by the injection! The patients who received this for the relief of pain all volunteered the statement that the respite from suffering more than compensated for the transient pain of injection. This procedure has been employed on more than twenty occasions without any evidence of necrosis. The injection, however, should be given deeply into the gluteus medius to avoid sloughing.

In the hope of avoiding the pain of intramuscular injection, the intravenous route has been employed in a few cases. A patient[5] suffering terrific pain from a coronary thrombosis was given 5 cc. of undiluted paraldehyde into the vein at the rate of about 1 cc. in three seconds. The patient lost consciousness in about ten seconds. Coincident with the onset of anesthesia was a slight amount of pharyngeal irritation accompanied by coughing and a strong smell of paraldehyde on the breath. After a few coughs the patient appeared completely relaxed and slept quietly for about an hour. Upon awakening the pain was much less severe and

could be adequately controlled with morphine.

Intravenous paraldehyde in 7 to 10 cc. doses has also been used to control the restlessness and headache of a hypertensive patient with evidence of extensive cerebro-vascular pathology. This patient[6] was violently nauseated by morphine and was unable to retain the barbituric acid derivatives by mouth or rectum. Quiet sleep was induced on each of six successive nights by the intravenous administration of 7 cc. of paraldehyde. The sleep so induced would last several hours but could be greatly prolonged by the rectal injection of sodium amytal Gr. V-X which was retained if given after consciousness had been lost. It is interesting to note that this patient began to show some degree of tolerance to paraldehyde after two or three injections, as there developed a latent period of ten to fifteen minutes between the injection and the onset of deep sleep. Such cases are probably not proper subjects for this treatment as their pain is hardly severe enough to warrant anesthesia and the relief from minor symptoms is too transient to be of much help in the long run. In this particular case intravenous paraldehyde was used because we had apparently exhausted every other method of giving the patient quiet sleep. When this type of patient reaches the convulsive stage, however, parenteral paraldehyde is the treatment of choice as its action is swift and an anesthetic dose is well within the limits of safety. Here it is often superior to morphine, for many of these patients have developed a degree of tolerance which makes difficult the estimation of a dose which will be immediately effective yet safe.

Tolerance to paraldehyde unquestionably develops and habit formation has been reported, but these should not be problems if the drug is used only occasionally to control severe pain or convulsive states requiring rapid anesthesia.

It must be admitted that paraldehyde, however administered, is unpleasant and for that reason it is not the first choice for the relief of pain. But it is cheap, stable, easy to procure, requires no sterilizing and has a wide margin of safety while being almost instantaneous in action when administered parenterally. This makes it an exceedingly useful drug for the control of convulsive states and for the relief of pain which is unaffected by morphine.

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5 Acknowledgment is made to Dr. Bernard Rabinovitz for permission to report this case.
6 Acknowledgment is made to Dr. Henry Ritter for permission to report this case from the medical service of the Springfield Hospital.

FOREIGN PROTEIN SENSITIZATION WITH MENINGEAL INVOLVEMENT DUE TO THE USE OF VACCINE*

BY WILLIAM E. HALL, M.D.,† AND THOMAS P. MURDOCK, M.D.†

FOREIGN protein sensitization with involvement of the meninges has not been dealt with extensively in American literature. The clinical picture is alarming. We feel that the disease occurs more frequently than has been believed, and because of this the importance of the condition should be stressed.

Swift and Boots[1] go so far as to say that most patients receiving considerable amounts of therapeutic serum, develop serum disease. If this is so, and certainly there seems to be sufficient evidence to support it, the condition warrants more than passing notice. The present tendency toward subcutaneous, intramuscular, and intravenous injections of foreign proteins will in all probability increase the frequency of protein sensitivity, and also the more serious meningeal complications. Serum disease seems much more likely to follow the use of scarlet fever antistreptococcus serum, than any of the other serums.

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In 1926 Kennedy[2] reported severe symptoms referable to the central nervous system in three patients with angioneurotic edema, which he attributed to a focal edema of the brain. In 1928[3] he also reported several cases of involvement of the nervous system, and one case showing meningeal signs and symptoms following administration of serum. He suggested two possibilities as causative factors: The toxicity of the serum, or the urticarial edema of the perineural tissue.

Sheppe[4] reported a case of meningitis in a laboratory technician who had been infected with meningococci while at work in the laboratory. The patient was given antimeningococcus serum intraspinally and intravenously. Subsequently the patient got well, and the spinal fluid sterile. This was followed by meningeal symptoms with sterile spinal fluid, and a cell count of 4536. He concluded that the final symptoms and signs were due to aseptic meningitis caused by the serum.

There is still another possibility of developing so-called aseptic meningitis and that is as the result of lumbar puncture. Hurxthal[5] recently

called this to the attention of the profession. He mentioned the increased frequency of lumbar puncture as a diagnostic procedure, and also the increased frequency of its use in spinal anesthesia. He believes that because of these, the possibilities of developing this disease are definitely increased.

Goldman[6] was stimulated to investigate aseptic meningitis because of the frequency with which the condition was found at autopsy. He showed that the condition occurred with great regularity in experimental animals that had received intraspinal injections of serum. Of course the question may here arise as to whether the condition is due in these cases to trauma to the meninges, or to the administration of serum.

Wilson and Hadden[7] have recently written on the question of neuritis and multiple neuritis following serum therapy. They reviewed the literature extensively, and reported several cases.

The accepted procedure in patients with suspected meningococcic meningitis has been to administer serum intraspinally and intravenously following the withdrawal of spinal fluid. In the light of the present case, we feel that this should be done only very cautiously, and only after a definite diagnosis has been made. In other words, we feel that serum should be administered intraspinally and intravenously, only in those cases in which the diagnosis is clear. In doubtful cases, the fluid should first be examined and if found positive, the serum is injected.

A thorough search of the available literature does not reveal a case of foreign protein sensitization with meningeal involvement, due to the use of a vaccine. That it does occur seems a certainty. The probability is that the sensitization is due to the foreign protein in the bodies of the bacteria. We feel that the use of vaccines in medicine has been increased to such an extent that the danger should be called to the attention of the profession. It is quite probable that many physicians, for one cause or another, frequently inject vaccines.

The accepted uses of vaccines for atrophic arthritis and pertussis, the uses of autogenous vaccines for various conditions, and the unscientific use of various kinds of stock vaccines for almost every infectious condition, mean that almost certainly there will be an increasing number of cases of foreign protein sensitization, and with these a certain number of cases with meningeal involvement.

All of the cases (except that reported below) that we have found in the literature and have reviewed, were associated with the use of serum, and most of the cases with the use of serum intraspinally whereas the case reported here was not associated with either.

We believe that the danger of developing serum disease is increased in children who have had diphtheria toxin-antitoxin, and because of this, the danger of serum therapy is markedly increased. The use of toxoid in the future will, in all probability, lessen the number of cases of serum disease.

The condition is difficult to diagnose, particularly if the skin manifestations are not prominent. The difficulties are increased if, as is frequently the case, the patient is not clear mentally, and it is impossible to obtain a clear history. Given semi-consciousness, headache, opisthotonos, fever, vomiting, positive Kernig, and discrete spots on the skin, one most naturally assumes that the condition is meningitis, and the probable clinical diagnosis is epidemic cerebrospinal meningitis. The spinal fluid is under increased pressure and is turbid. (The injection of serum at that point adds insult to injury.) We feel that if there is any doubt as to the diagnosis, treatment should be postponed until the spinal fluid has been examined.

We think there are two points worthy of mention as to differential diagnosis: first, the patients do not look so ill as the findings or signs would indicate; secondly, the low leucocyte count is not in conformity with the clinical picture of epidemic meningitis. It is now generally accepted that at the onset of symptoms due to foreign protein sensitization, there is leukopenia. Later on it is common to find a leukocytosis. We have the feeling that many cases of meningeal involvement due to foreign protein sensitization have gone unrecognized because of the acceptance of the syndrome as generally outlined; that is, prostration, fever, adenopathy, urticaria, painful joints, and vomiting following the administration of a foreign protein by injection. Meningeal involvement not being sought for or expected, is overlooked.

CASE REPORT

H. K., a white female patient, aged 43 years, was admitted to the Hospital on April 3, 1933.

C. C. Headache and vomiting.

P. H. The past history disclosed the usual children's diseases without complications. Late in 1931 she had an attack of furunculosis lasting several weeks. In 1932 she was ill with "rheumatism" for several weeks. She had recurrence of this for a few weeks prior to admission to the hospital. There was no past history of hayfever or previous urticaria.

F. H. Mother living and well. Father dead, cause unknown. One sister living and well; one brother has sinus trouble.

P. I. The onset of the present illness occurred suddenly on the day before admission with headache, vomiting, and backache. These symptoms persisted all day when one of us (W. E. H.) was called to see her. A clear history was impossible because the patient was semiconscious. The temperature was 102°, pulse 100, respirations 30.

P. E. Pupils equal and react to light and accommodation. Conjunctivae and sclerae injected. Moderate edema of both eyelids. No extraocular palsies. Tongue moist. Teeth in good con-

dition. Pharynx clear. Neck stiff. No tenderness over sinuses. No facial palsies. Thyroid not enlarged. Cervical glands not enlarged. Lungs negative. Heart not enlarged, sounds of fair quality, systolic murmur over the mitral area. Pulmonic second sound was booming, and greater than the aortic second sound. Bl. Pr. 145/85. Liver and spleen could not be felt. There were no tender points and no masses. There was an old scar in the right lower quadrant. Pelvic and rectal examinations were negative. There were prominent veins in the left leg. The right knee jerk was hyperactive. Babinski negative. On her back, chest, abdomen, and over the backs of her hands and wrists were sharply defined red spots, varying in size from one centimeter to three centimeters in diameter. These were surrounded by a narrow ridge of grey, and faded on pressure.

A diagnosis of meningitis was made, and she was admitted to the Meriden Hospital. A lumbar puncture was done. The fluid was slightly cloudy, and under increased pressure. She was given antimeningococcic serum intraspinally. Examination of the spinal fluid showed the total cell count to be 6,804, polymorphonuclears 89 per cent, small lymphocytes 6 per cent, large lymphocytes 5 per cent. No organisms were found on smear or culture. Globulin 4 plus. No reduction of sugar. Colloidal gold curve 1244445533 (meningitic curve). Wassermann negative.

Following this the patient improved rapidly. On the fourth she still complained of some headache, but this was not so severe. The temperature on the evening of April 4, 1933 was 99.8° rectally, pulse 100, respirations 20. From then on until discharge the vital signs were in normal range.

On April 4, 1933 it was possible to obtain a clear history from the patient. She stated that she had been taking some injections for "rheumatism". The doctor informed us that she had been given several injections of antistreptococcic vaccine for her arthritis; the last injection was 1 cc. and had been given on the day preceding the onset of her present illness. We also learned that she had an attack of urticaria following vaccine injection one month ago.

On April 4, 1933 lumbar puncture was repeated. The total cell count was 4740, polymorphonuclears 88 per cent. No organisms were found on smear or culture.

The urticaria had practically disappeared on April 5, 1933, two days following admission.

On April 5, 1933 lumbar puncture was repeated. The cell count was 1620, and polymorphonuclears 86 per cent.

The laboratory findings were R.B.C. 4,040,000, W.B.C. 6,000, Hemoglobin (Tallqvist's) 80 per cent, polymorphonuclears 71 per cent; small lymphocytes 19 per cent, large lymphocytes 10 per cent, filamented 84 per cent, nonfilamented 16 per cent. Blood Wassermann negative. Non-protein nitrogen 40 mgs. Blood sugar 136 mgs. Blood culture negative.

The patient continued to improve and was discharged on April 10, 1933, one week after admission. Two days later on April 12, 1933 the patient complained of feeling weak, of smarting of the eyes, and of vague pains. On the next day, April 13, 1933, she was readmitted to the hospital. On admission the temperature was 99°, pulse 100, and respirations 20.

There were a few urticarial spots on her back and chest, and slight edema of her eyelids; otherwise the findings were essentially the same as on the previous admission. A spinal tap was done, and the total cell count was 564, with 77 per cent polymorphonuclears. The fluid was under increased pressure, and was slightly turbid. On April 15, 1933 the rash involved her arms, forearms, legs, chest and back, and itched furiously. A dermatological consultation was requested, and Dr. R. V. Quinlan saw her and concurred in the diagnosis. Histamine phosphate was used at this time for the urticaria. This was followed by local relief. On April 17, 1933 the rash was still present, but not so marked, and she developed some painful, red and swollen joints involving her wrists and great toes. This gradually subsided, and from then on her convalescence was uneventful. A lumbar puncture was done on April 21, 1933 and showed a cell count of 166, with polymorphonuclears 56 per cent, and lymphocytes 44 per cent. Smear and culture were negative. The patient was discharged on April 25, 1933.

It is our feeling that the recurrence in this patient was due to the administration of antimeningococcic serum before the serological work had been completed and the diagnosis established.

That the original attack was caused by streptococcic vaccine seems certain. This opinion is supported by the fact that a history was obtained of a previous attack of urticaria one month before the present attack, and that it followed an injection of streptococcic vaccine.

Of particular interest is the extremely high cell count, ranging from 6804 to 4740, to 1620, to 564, and finally to 166.

CONCLUSIONS

1. The literature on meningeal involvement due to foreign protein sensitization has been reviewed.
2. The disease can be caused by vaccines.
3. This warning is issued because of the increased frequency of the use of serums and vaccines.

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RECURRENT DISLOCATION OF THE SHOULDER

BY FREDERIC JAY COTTON, M.D.,‡ AND GORDON MACKAY MORRISON, M.D.‡

RECURRENT anterior (subcoracoid) dislocation of the shoulder is a not unusual sequel of reduced traumatic dislocation. It occurs in young strong active men, usually, and urgently calls for relief. A large number of operations for cure have been devised, not a few of which have excellent records as to end results —so far as checking reluxation is concerned*.

This note is a plea for a really *restorative, reconstructive* operation that sacrifices no structure, no function, and really reproduces original function and power. It seems to be at least as effective as any other, when performed as outlined.

The essential or at least constant lesions found† are the general slack of the whole capsule, particularly in front toward the axilla, and, associated with this, a retraction of the subscapularis muscle.

It is futile to talk of anything that has to do with the *tendon* of the subscapularis.

There is no such thing. That is to say, no isolated tendon structures, differentiated from the capsule into which the muscle is inserted.

The *muscle* is inserted on the front face of the thick fibrous capsule, normally within an inch of the glenoid edge; in these cases of recurrent luxation it may be found retracted not less than two and one-half inches toward the midline, to be found only by deep retraction, sometimes only after strong dragging outward with heavy double hooks on the V-shaped portion of the fibrous capsule, cut and loosened for advancement. (See figure, A, 8.)

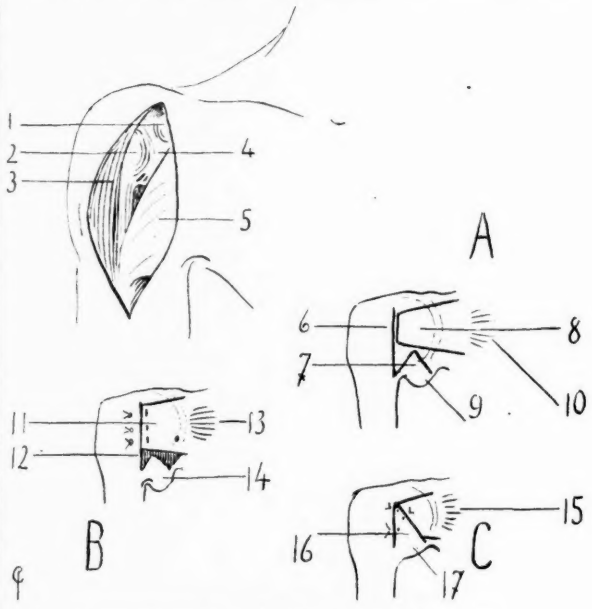
Oddly enough, the muscle once found is normal looking, not evidently atrophic.

The essence of the operation here described is an advancement of the muscle to its normal relation to the joint, combined with a reefing of the loose capsule in front and below.

The operation began as an anterior capsulorrhaphy devised and shown by the late Dr. Herbert Leslie Burrell.

This alone proved not reliably efficient, and there have been added a reefing upward and forward of the loose capsule in the axilla,—and, more particularly, a really anatomical dissection of that part of the capsule into which the subscapularis muscle should be inserted, and an advancement of this muscle, often through one and one-half inches to its proper anatomical place. So modified, the operation works. The approach is by the familiar Hueter incision, from an inch above the coracoid process down along the inner edge of the deltoid to an inch below the insertion of the great pectoral.

The deep approach is between the deltoid and the clavicular portion of the pectoral. In this interval lies the cephalic vein, which may be retracted inward with the pectoral. It is often convenient to cut the upper half of the tendon of the pectoralis major. This gives much more room, and does no harm. After the great muscles are once separated, one comes directly onto the capsule. Rotate the arm outward and the head presents. Further procedure may best be understood from the drawings. The flap that is to include the subscapularis muscle goes through



RECURRENT DISLOCATION OF SHOULDER.

UPPER LEFT.

1. Coracoid process.
2. Head of humerus.
3. Deltoid muscle (and cephalic vein).
4. Edge of glenoid cavity.
5. Pectoralis major muscle.

FIG. A.

6. Vertical cut in capsule, internal to bicipital groove.
7. Triangular flap of capsule (to take up slack capsule in the axilla).
8. Flap to include the subscapularis insertion.
9. Lax capsule in axilla.
10. Subscapularis muscle.

FIG. B.

11. Shortened flap carried outward taking with it the subscapular muscle.
12. Redundant capsule, trimmed out.
13. Subscapular muscle.
14. Lax capsule in axilla.

FIG. C.

15. Advanced subscapularis muscle.
16. Triangle (9 of fig. A) brought upward and sutured in place.
17. Axillary portion of the capsule pulled up, and made flat.

*Turner, Thomas, Clairmont, Kellogg Speed, Oudard, Henderson, Nicola, Roberts, et al.

†We have almost never—only once to be exact—found even a trace of glenoid defect, though no doubt it does occur.

‡Cotton and Morrison—For records and addresses of authors see page 817, issue of April 12, 1934.

the fibrous capsule to the synovial capsule, which is at this place, a separate layer.

The whole operation can be done without opening the joint. Any opening that may be made into joint or bursa is of no consequence whatever. We have never taken out any part of the undoubtedly redundant synovial layer, nor have I seen reason to do so. Suturing of the shortened V-shaped flap, carrying the muscle, to the cut edge of capsule near the biceps groove must be very solid; it must withstand some strain. Chromic gut No. 2 is good. There is no room to use fascia. The V brought up from underneath out of the axilla is trimmed and used as a covering flap. It is under no tension—suturing with No. 1 chromic gut is ample.

The mechanical part of the operation done, the wound closes itself. To control undue dilatation of the stripped cephalic vein one may bury it between the great muscles and lightly suture them about it. The cut pectoral insertion needs no sutures. The arm is put up with elbow at side, and the forearm across at the waist. Pressure dressing is not feasible. A light soft sandbag over the front of the shoulder for from 12 to 24 hours is advantageous, and usually well tolerated.

Motion begins at three weeks, and useful motion with solid healing follows within ten weeks at most. There is always, if the operation is properly and solidly done, some limitation of abduction and outward rotation for from four to six months*.

CASE 1. R. W. M., male, aged 27. In Public Health Service. Seen March 21, 1920. Dislocated left shoulder at bayonet practice in September, 1917, and a week or so later from a slight backward fall. Discharged from the army, December, 1917. While at home the shoulder was out four or five times. Redrafted September, 1918. Discharged again in May, 1919, for same disability. Shoulder (left), has been dislocated seventeen times in all, despite an operation at the Boston City Hospital in 1918, and two operations in the army. Six weeks ago the right shoulder went out while the doctor was reducing the left shoulder. The right has been out three times in all. General musculature poor; about both shoulders and *scapulae* very poor. A neurologist fails to confirm the suspicion of progressive muscular atrophy. The left shoulder is a mess of scars, front and back. Admitted to the Parker Hill Hospital, April 12, 1920. Operated on the left shoulder Aprii 16, 1920. (F. J. C.) Usual anterior incision. Typical Burrell-Cotton operation with capsule reefing and advancement of subscapular muscle. All musculature is poor.

Operated elsewhere on the right side twice in 1921 with recurrence.

Examination: March 24, 1922. Left shoulder shows muscle redeveloped, practically normal. Right shoulder has limited motion and two scars. Subluxation rather than total dislocation. Operated at City Hospital 1922 (F. J. C.) for right shoulder. Excision of scar, type Burrell-Cotton operation on right shoulder. Seen July, 1933. Not very good musculature about shoulders even now, and some limitation of motion on right. There has been no reluxation of either shoulder after the muscle capsule reefing.

CASE 2. V. R., female, aged 24. Seen September 7, 1929. Hospital technician. She was thrown from a "cutter" eight years ago, and dislocated her left shoulder. Luxation since then five times needing reduction; five other times partially dislocated. Joint has an unsafe feeling, and is sometimes painful. Operation recommended. Finally came to operation, June 29, 1930 at the Boston City Hospital (F. J. C.). Type Burrell-Cotton operation. Normal recovery. Examined June, 1933. Shoulder in normal shape, in all ways. Has had no recurrence.

CASE 3. E. F. C., male, aged 23. Wireman in an electric corporation. Seen October 19, 1932, referred by the late Dr. John F. Sweet of Newport, Rhode Island.

On October 17, 1931, he reported that he injured his left shoulder, cranking a truck about five years ago (dates are vague), and it has "gone out ten or twelve times a year" since then. The last time six days ago, Dr. Sweet had to put it back, and sent him to Boston.

He proved to be a young Frenchman of admirable build and amazing muscle. The shoulder showed nothing, but was a little sore.

Operation at the Faulkner Hospital, October 28, 1932. (Cotton and Morrison.) Usual incision and approach. Marked looseness of capsule, in front only. Subscapularis much retracted. Usual excision and reefing done—taking up muscle and capsule slack of near two inches. Six double No. 2 chromic gut sutures to hold it, in view of the enormous muscles. Sutures through muscles to control dilatation of huge cephalic vein. No reefing of axillary part of capsule.

Motion begun early, and from December 15th on he was allowed full and free use of it.

He was last seen August 25, 1933. He has been using the arm continuously, but only within a fortnight has he had absolutely full upward motion. Now 100 per cent motion and use. The deltoid muscle is nearly as huge as on the right. No sign of recurrence. In a young giant like this it pays to quilt the capsule liberally, and solidly, but there is, for a time, for this very reason, some resulting loss of motion.

CASE 4. H. G., male. Original injury two years before. "Out" several times, most recently while swimming. He demands relief. Admitted September 11, 1928. Discharged September 22, 1928. Operation September 13, 1928 at the Boston City Hospital.

Usual aseptic precautions. Anterolateral incision over left shoulder. Cephalic vein located. The deltoid was separated from the pectoralis major and retracted with the cephalic vein. The shoulder capsule was exposed, a reef taken in the capsule and the subscapular tendon shortened over the capsule. No recurrence to date. Examination July, 1933.

CASE 5. G. McL., aged 22, florist, seen May 23, 1933, referred by Dr. Robert J. Graves of Concord, N. H. No history of original trauma, but for seven or eight years, about once a year, the right shoulder has been "throwing out". Ten days ago Dr. Graves had to reduce it. Examination showed a vigorous young man, with only local difficulty. Operation June 2, 1933 at the Faulkner Hospital. The usual incision and technic. The subscapularis was advanced about an inch and a half and moored

*Advantageous to clear this last hindrance is the *free "pendulum" exercise, by the patient*, who lies on a bed, face down, with the arm hanging over the edge, and a bit later, the swinging of light Indian clubs.

with No. 2 chromic gut. The lower capsule in the axilla was not lax, but caught up in a V, very snugly, according to the usual technic.

Motion from June 26, 1933. On July 6, 1933, set about vigorous limbering up exercises. A prognosis of six months before regaining absolutely normal abduction and outward rotation was given. Discharged July 6, 1933. No recurrence as yet.

Case 6. M. K., female, aged 35. Seen March 17, 1927. An industrial case, completely disabled from recurring subcoracoid dislocation of the shoulder, often "out" with frequent subluxations in the intervals.

Operated on April 13, 1927 at the Faulkner Hospital with Dr. Henry Marble. Usual approach. The capsule proved unusually "baggy" in the axilla. The subscapular muscle was broad, flat, soft, displaced inward and downward, but easily drawn into the wound. The usual operation was varied by using a large inverted V cut in the capsule, enabling a shortening and raising of the subscapularis to be combined with an unusual pulling up of the capsule from below. The V flap was liberally trimmed, brought up in front and sutured as usual.

Healing was prompt, but limbering up of the joint went very slowly, perhaps partly because she lived 30 miles from Boston.

There was long persistent muscle spasm, and obviously a neurotic element in the case, with varying disability.

Up to November 3, 1928, however, there had been no recurrence of luxation or subluxation.

MEDICAL PROGRESS

PROGRESS IN THE DIAGNOSIS AND TREATMENT OF SYPHILIS, 1933

BY AUSTIN W. CHEEVER, M.D.*

THERE have been a few years that were epochal in the history of the treatment of syphilis: 1905, with the discovery of the spirochete; 1906, with the development of the Wassermann reaction as a practical test for syphilis; 1910, with the success of Ehrlich's six hundred and sixth experiment; and a few others. With these exceptions, the progress in any one year has not seemed consequential, but our small advances have been cumulative and over a period of years they show a long step forward as C. Morton Smith[1] makes clear in his review of the changes in the treatment of syphilis during the last twenty years. He traces our gain in the relief and control of this disease and in our efforts to educate the public in its attitude toward the syphilitic. One proof of our success in the latter respect is evidenced by the fact that the past year in Boston saw what an officer of the Massachusetts Society for Social Hygiene[2] hailed as "the most recent single event of importance in the field of social hygiene". It was the production of the picture "Damaged Lives" which played to crowded houses. Equally gratifying was the fact that Boston dramatic critics dared to refer to the keynote of the play, gonorrhea and syphilis, by name without resorting to euphemisms. In this same city Nelson of the State Department of Public Health[3] has been giving radio broadcasts on syphilis and gonorrhea with, it is said, not a single complaint against their frankness but a number of favorable comments and requests for further information. At least one newspaper printed an editorial commending the talks.

However, there still remains much educational work to be done when a court[4] will annul a marriage because a woman having had advanced syphilis before marriage did not disclose the information to her husband. With the same lack of understanding the appellate court, Louisiana,[5] has ruled that an insurance policy which excludes from its coverage venereal diseases imposes no obligation on the insurer to compensate for the effects of syphilis, even though innocently acquired.

If we are to prevent blindness from interstitial keratitis and the mental and physical defects of congenital syphilis, the medical public must be educated to greater vigilance in the care of the pregnant mother. Lees[6] reports that in Edinburgh among 1000 untreated pregnancies only 24.5 per cent of the children were sufficiently healthy to survive while a similar survey of efficiently treated mothers showed 89.2 per cent of healthy children, and an even larger percentage, if the treatment of the mother was begun before the sixth month of pregnancy. At the Memphis General Hospital[7] 1000 cases of syphilis in pregnant women were discovered over a period of ten years. Of those receiving treatment, 65 per cent had normal, full-term babies with no evidence of syphilis. Of those receiving no treatment, none had a normal, full-term infant with no serological or clinical evidence. In view of these reports it is encouraging to know that the American Medical Association at its annual meeting in 1933 adopted a resolution[8] requesting the House of Delegates to appoint a committee to arrange methods by which coöperation might be secured with the American Medical Association, the National Society for Prevention of Blindness, the American Social Hygiene Association, the obstetric and ophthalmologic societies, the American Dermatological Association, and public health organizations and such others as can help, to see that blood examinations are made of all pregnant women so that all cases of syphilis may be treated.

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The use of artificially produced heat in the treatment of central nervous system syphilis and the favorable results from malaria have stimulated many investigators to try to devise less dangerous and more controllable methods of producing fever. M. O. Nelson[9] recommends combined typhoid vaccine which he found brought improvement in patients with resistant neurosyphilis. Epstein and Paul[10] for two years have been using a diathermy apparatus capable of raising the body temperature to 41.1° (106° F.). They describe their technic and results on a group of twenty-eight patients with various forms of central nervous system syphilis. Clinical improvement was noted in patients with all types of the disease but especially so in those with tabes dorsalis. One fatality occurred in a patient of advanced age with marked mental deterioration. Graham[11] obtained results better than those from malaria, he thinks, in twenty-four cases treated by diathermy, though he records one death due to respiratory failure attributable to treatment. Worthing[12] describes a diathermy machine in which, so far, only a small number of patients have been treated. His results agree in general with those of other workers, but the author emphasizes the extreme discomfort of the patient after the temperature rises above 102 degrees.

Freeman, Fong, and Rosenberg[13], on the other hand, found the substitution of diathermy for malaria in the treatment of dementia paralytica far from promising. They feel that early reports on the use of mechanical measures for producing fever have been premature. O'Leary[14], too, feels that the results from malaria are still superior to those obtained from electrical machines. He has observed over a period of eight years almost one thousand patients with neurosyphilis who were treated with malaria and he has found that in more than three-fourths of the cases of asymptomatic dementia paralytica, clinical progress of the disease was arrested, and the serologic tests of the spinal fluid became reversed to complete negativity in almost half the cases of asymptomatic neurosyphilis in which routine measures of treatment had previously failed to influence the serology. The results in the treatment of tabes dorsalis and of the tabetic form of dementia paralytica were likewise encouraging.

In 1932 Gennerich[15] reported on a new mercury preparation which he found intensely effective for all stages of syphilis but less active than arsphenamine. This year Lawless[16] reports on this drug, colloidal mercuric sulphide. He found it painless with no palpable infiltration and decidedly effective though too slow to be of use in the infectious stages of syphilis. It is highly recommended as a supportive drug with the arsphenamines and bismuth. A serious drawback is its rather constant, resistant staining of the skin at the site of injection.

Because of inconclusive clinical results with 50 per cent mild mercurous chloride inunctions, Cole et al[17] made a study of the mercury excretions in order to obtain a better criterion of its true absorption from the skin. They found it very poorly absorbed.

After a study of the different types of bismuth preparations: watery solutions, a solution in ethylene glycol, oily solutions and oil suspensions, Sollmann, Cole, and Henderson[18] decided that the total dosage is much more important than the particular preparation used. They were impressed by the similarity in the absorption and excretion rate of all these compounds except the oil suspension of bismuth salicylate which showed much slower absorption and excretion. Distinct doubt is thrown upon the desirability of too rapid absorption in bismuth because of a correspondingly rapid excretion rate and the accompanying tendency to overburden the kidneys and liver. After studying five typical bismuth preparations as to rate of absorption, distribution in kidneys and liver, urinary excretion, irritation at site of injection, and toxicity, Thompson et al[19] show that it is not yet established that a rapid absorption rate will result in a more effective concentration against syphilis. They confirmed the intimate relationship between absorption rate and toxicity, the preparations showing fastest absorption being most toxic.

An excellent summary of our knowledge of bismuth at the present time is found in a discussion of bismuth compounds by Irgang, Alexander, and Sala[20].

Bismuth has been recommended as a prophylactic against syphilis, and in this connection it is interesting to see the report by Zetterholm[21] of a reinfection in a patient during the course of continuous bismuth treatment. The same situation holds for stovarsol by mouth which has been recommended as a prophylactic. Tzanck et al[22] report an instance of a patient's developing syphilis while taking stovarsol by mouth for non-syphilitic lesions.

After studying the effects of arsphenamine and tryparsamide in systemic syphilis and neurosyphilis in sodoku and in malaria, Solomon, Epstein, and Berk[23] conclude that the value of the former drug is in its spirocheticidal and plasmodicidal effect as indicated by its action in stopping fever in malaria and in clearing the blood of plasmodia in inoculation malaria, while tryparsamide showed no effect on either fever or blood. However, this latter drug shows a power to stimulate tissue to rapid repair, a local action within the local areas of the nervous system.

Raiziss and Severac[24] made a very careful comparison of bismarsen and arsphenamine in rabbit syphilis. They conclude that bismarsen is as effective as arsphenamine in experimental syphilis in rabbits.

A comparative study of the results and reactions of neoarsphenamine and arsphenamine was made by Thornley[25]. Taking a group of 999 patients who had been under observation or treatment for six months or longer, he found the difference in efficacy between the two drugs not very great when the neoarsphenamine is given well diluted, but when for greater convenience it is given in concentration, the reactions are much more numerous than with arsphenamine. To lessen the chances of treating syphilis with a brand of neoarsphenamine below the average in curative activity, since the different brands vary in anti-syphilitic properties, Schamberg and Kolmer[26] urge that the National Institute of Health establish official standards of therapeutic efficiency for this drug in addition to their present tests for toxicity.

A method of treatment worthy of further study is the work being done by Wittenberg[27] on a plan to introduce larger amounts of the arsphenamines into the central nervous system. His idea is to use drugs of the epinephrine group to cause cerebral vasodilation, followed in two minutes, the time of the height of the dilation, by neoarsphenamine.

A distinct inhibitory effect on syphilis in rabbits through the use of trypan blue has been found by Morgan et al[28]. They suggest that it is produced not by spirocheticidal action but by the increase of the number and activity of the phagocytic mononuclear cells.

Robinson[29] discusses a case of "fixed" dermatitis in a patient having tryparsamide showing a striking resemblance to that caused by phenolphthalein. The eruption could be brought out at will by giving injections of tryparsamide.

Serefis[30] believes that salvarsan intolerance can be prevented or cured by decreasing the sodium chloride content of the food and increasing its content of potassium, calcium, and magnesium.

Belding and Arrowood[31] made a quantitative study of three flocculation tests, Sigma, Kahn, and Hinton, from the standpoint of sensitiveness and variability. They found the Hinton test the most sensitive in detecting syphilitic reagin, the Kahn next, and the Sigma the least. The Hinton showed less variability, they report, than the other two. These investigators[32] in making preliminary experiments to determine the suitability of the Hinton test for laboratory routine found what they consider possibilities for adapting this test for quantitative work. They described the technical considerations involved in such a modification.

Dental observations are being made on syphilitic teeth by induced electric currents. So far only a preliminary report[33] has been made of abnormalities which seem to be connected with syphilis.

Wile[34] has made a very careful analysis of a group of 157 cases of syphilis with cardiovascular involvement occurring preponderantly in males averaging from 46 to 53 years in age, the milder involvement being at the later age. The average duration of the disease before clinical detection varied from 15 years in the severe cases to 24 in the mild. A moderate number of the group had had some previous antisyphilitic treatment, but practically none had had what would now be called satisfactory.

The pathology, clinical features, and treatment of cardiovascular syphilis have been most ably presented by Mallory, White, and Smith[35] and should be read in their entirety by those interested in the problem.

After examining 1,548 patients with cancer of the mouth at the Collis P. Huntington Memorial Hospital, Lund[36] found that 122 had syphilis, also. Of 401 patients with cancer of the tongue, 70 had syphilis. Lund calls syphilis a contributing cause of cancer of the tongue. In the experience of Molesworth[37], who reported last year on this subject, 20 to 30 per cent of cancers in the mouth occurred in persons showing positive serology.

In the opinion of Wheeler[38] a persistently positive serum reaction means uncured syphilis. He arrived at this conclusion after a study of 200 patients with persistently positive serology.

Herrmann[39] feels that syphilitic disease of the peripheral arteries is not an uncommon occurrence. He describes three types: the angiospastic, occurring in upper extremities and manifesting itself as a chronic arteriospasm; the endarteritic, seen in the legs and feet with constant pain; the thromboarteritic, uncommon, manifesting itself by extensive organic occlusion of all the major pathways of the extremities without evidence of gross gangrene. Active antisyphilitic treatment should be the first step in the management of these disturbances. Treatment by means of an intermittent negative pressure environment is the most effective in the opinion of the author.

At an Illinois State Institution[40] where up to 1930 there was no routine antisyphilitic treatment, records of all patients up to that time were carefully examined, and of the 59 found to have positive reactions on admission, eighteen (30.5 per cent) of varying ages and length of stay now have negative tests although they have received no antisyphilitic treatment.

It is not yet sufficiently realized that intraurethral chancres occur more frequently than is generally supposed. Strachstein[41] reports six cases and calls attention to the fact that a persistent so-called simple urethritis may indicate a probable intraurethral chancre. Serra[42] reviews the cases of extragenital primaries seen at the dermatosyphilographic clinic of Turin from 1889-1931. Nine and two-tenths per cent of the total number of cases showed extragenital lesions. Cases of extragenital syphilitic infec-

tions in six female members of one family transmitted through the common use of eating utensils is reported by Jenö Follman[43].

It is not possible to overemphasize the importance of care in choosing transfusion donors. A secondary rash developed in a girl fourteen years of age after she had received blood from her brother, whose chancre appeared four or five days later[44].

For years it has been a controversial point as to whether there are other forms of the organisms of syphilis than the familiar Spirocheta pallida. Numerous facts can be satisfactorily explained on the ground of a life cycle such as is known in some other organisms. Levaditi and others[45] working at the Pasteur Institute in Paris are studying this problem. They feel that it is probable that the virus undergoes certain phases in its evolutionary cycle before it can invade the central nervous system and that it then passes through alternating phases in the nervous system, sometimes infravisible, sometimes in the form of spirochetes, and that this explains the inconstancy of the findings in the cortex of general paralytics. Coutts[46] believes that certain cases of syphilitic infection with no apparent infective lesions can be explained by the possibility that latent syphilitics may become active carriers of Spirocheta pallida. He cites two cases where this seems the only satisfactory explanation as to the mode of contagion.

If we are to gain complete control of syphilis, sources of infection and exposures must be traced. Smith and Brumfield[47] by the careful questioning of 119 patients with rather recent syphilis discovered and placed under treatment 93 syphilitic contacts. From one original case 19 contacts were traced, having either primary or secondary syphilis, or positive blood tests. Nelson of the Massachusetts State Department of Public Health[48] feels that much will be accomplished when the physician in his office practice uses the same careful follow-up that is used in the best clinics. He describes an experimental service which the State has offered a few physicians treating syphilis and gonorrhea whereby a trained investigator or social worker is sent out by the State Department to work with the doctor on lapsed cases. Though the results were sufficient to demonstrate the value of such service, it has not yet been possible to "sell" this service to the majority of physicians.

A study of field work over a period of nine months in the clinics and activities of seven institutions has been made by Morris[49]. The author summarizes her findings and makes general recommendations. Her report is of interest to institutional workers.

Two valuable books have added to the year's progress. The History and Epidemiology of Syphilis by William Allen Pusey[50] presents in an unusually attractive form the three Gehrmann lectures delivered by the author at the University of Illinois on the historical and epidemiological aspects of syphilis. Joseph Earle Moore[50] in the Modern Treatment of Syphilis has given us a monograph of over five hundred pages generously supplied with references to the best literature on the subject. By the very free use of graphs and tables and by employing the simplest language possible, this book makes readily available, information as to what can be done for patients with various types of syphilis and what results may be expected from the treatment.

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MORTALITY RATES

Telegraphic returns from 86 cities, with a total population of thirty-seven millions for the week ending April 28, indicate a mortality rate of 12.0 as against a rate of 11.3 for the corresponding week last year. The highest rate (18.4) appears for Washington, D. C., and the lowest (6.8) for Evansville, Ind., and Long Beach, Calif. The highest infant mortality rate (16.4) appears for San Antonio, Texas, and the lowest for Albany, N. Y., Fort Wayne, Ind., Long Beach, Calif., New Haven, Conn., Omaha, Neb., Somerville, Mass., South Bend, Ind., Spokane, Wash., Syracuse, N. Y., Tampa, Fla., Utica, N. Y., Waterbury, Conn., and Youngstown, Ohio, which reported no infant mortality.

The annual rate for 86 cities is 12.6 for the seventeen weeks of 1934 as against a rate of 12.0 for the corresponding period of the previous year.

SUMMARY OF DEATHS AND DEATH RATES (ANNUAL BASIS) FROM AUTOMOBILE ACCIDENTS PER 100,000 ESTIMATED POPULATION FOR 86 CITIES FOR CORRESPONDING PERIODS OF 1934 AND 1933.

	Week ending April 28, 1934	Week ending April 29, 1933	First 17 weeks 1934	First 17 weeks 1933
Total deaths	134	113	2,660	2,344
Death rate	18.7	15.7	21.8	19.2
Deaths due to accidents in city	104	90	2,170	1,892
Death rate	14.5	12.5	17.8	15.5

—*Bureau of the Census.*

MR. STRAWSON RESIGNS

After fifteen years with the National Tuberculosis Association, Arthur J. Strawson has resigned to accept the position of executive secretary of the Southern Worcester County Health Association, 5 Pleasant Street, Worcester, Massachusetts. He will assume his new office on June 4.

Upon graduating from Northwestern University in 1908, Mr. Strawson was with *The Survey* and in settlement work for three years and then spent four years with the Chicago Tuberculosis Institute and the Illinois Tuberculosis Association. He left Chicago to become executive secretary of the Indiana Tuberculosis Association, resigning from this position for work with the American Red Cross during the World War. He served in camps for the Red Cross in Kentucky and Indiana and later was a field agent for National Headquarters. In 1919 he became regional secretary for the National Tuberculosis Association in the North Mississippi Valley with headquarters in Indianapolis. When the regional offices were discontinued in 1920, he came to New York as field secretary.

Mr. Strawson's countrywide experience has familiarized him with all phases of the program for tuberculosis control. The staff of the National Association feel profound regret at his resignation but congratulate Worcester County on securing his valuable services and rejoice that he is to continue in the tuberculosis field.—*Bulletin of the National Tuberculosis Association.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 20201

PRESENTATION OF CASE

The patient, a fifty-eight year old Irish cook, was perfectly well until one week before entry, at which time she began to have loss of appetite. Five days before entry she had a rather severe upper abdominal pain which radiated through the body to both shoulder blades. Aspirin did not give relief. She began to vomit immediately after eating, despite the fact that she had been taking only soft solid foods. Two or three days before admission the pain became less severe, but the vomiting persisted, and at this time there was coffee grounds vomitus. This condition persisted until admission and produced increasing weakness.

Physical examination showed an obese middle-aged woman mentally alert and talkative. She did not appear ill except for a sallow complexion and the fact that she was vomiting coffee grounds material without much nausea or retching. The sclerae were muddy but not definitely icteric. The heart was moderately enlarged. The aortic second sound was loud. There was a systolic blowing murmur at the apex and the base. The blood pressure was 280/135. The abdomen was soft and not tender. The examination was brief because manipulation brought on further hematemesis. No organs or masses were noted.

Examination of the urine showed a yellow test for sugar and a positive acetone. There was no albumin. The blood sugar was 312 milligrams. The carbon dioxide combining power was 51+ volumes per cent. Examination of the blood showed a red cell count of 5,000,000, 75 per cent hemoglobin and a white cell count of 17,000.

The temperature was 96.4°, the pulse 160, the respiratory rate 25.

In the Emergency Ward she was immediately given intravenous glucose five per cent, followed by thirty units of insulin. When she arrived in the medical ward she went into further collapse. The temperature was 100°, the pulse 160 and the respiration 45. The blood pressure was 100/70. She was cold and sweating. A clysis of 2½ per cent glucose was given, and 500 cubic centimeters of citrated blood by transfusion. The patient felt somewhat better and stopped vomiting. The pulse fell to 130 and the blood pressure rose to 130/70. This improved condition lasted until seven o'clock the following morning. At that time her blood pressure fell to 60-70 systolic and the pulse became too feeble to count. A second transfusion of 500 cubic centimeters of citrated blood was given. That afternoon she vomited two more kidney basinfuls of coffee grounds material. The blood sugar was down to 114 milligrams. The carbon dioxide combining power was 50.7 volumes per cent. Her abdomen became fuller and more tense, but no free fluid was demonstrated. There was a diffuse tenderness all over the abdomen, with some spasm. She rapidly failed and died that day.

DIFFERENTIAL DIAGNOSIS

DR. OLIVER COPE: The essentials seem to me to be that this patient is a woman of fifty-eight years who, following a period of at least a week's duration of loss of appetite and vomiting, was found to have hypertension and was incriminated with diabetes.

The past history is lacking.

The essentials of the physical examination at the time of her entry are unavoidably casual because of the fact that the vomiting increased as a result of any manipulation.

In looking at the subsequent history the diabetes seems to me to be distinctly incidental, although apparently she was treated with that seriously in mind. I believe it was the kind of diabetes that might occur in a patient of her age with arteriosclerosis and a mild tendency to diabetes aggravated by starvation and vomiting. I therefore would rule out the diabetes and I would think of those things that might have increased her blood sugar level and caused the excretion of sugar in her urine. The first thing I think of is of course an infection, and I would rule out rarer things like bronzed diabetes. I do not think that a sallow complexion and muddy sclerae are enough to incriminate her in that regard.

When we consider her subsequent course during the hospital stay—first of all she was very talkative and alert, and then went into rapid collapse following only a week's history—I should think of peritonitis in spite of the fact that we are told that her abdomen showed no tenderness and no masses. Her symptoms are certainly consistent with peritonitis. They are, however, no more specific than upper abdominal discomfort and pain between both shoulder blades. Peritonitis in a woman of her age might arise from many different sources. The thing that one would want to know is what one could feel in her abdomen at the time one saw her. They felt nothing, therefore it comes down to probabilities. I should like to commit myself and say that the most probable thing is an acute appendicitis which ruptured. The picture fits into that diagnosis. She had loss of appetite for two days and then sudden onset of pain and vomiting.

I omitted to speak of the coffee grounds material. There is no mention that that was actual blood. If so it would make one think of malignancy. It does not seem to me to fit into the picture of bleeding ulcer or of an uncomplicated intestinal obstruction. You could possibly get blood under the latter circumstance. I am still inclined, even though there was a positive guaiac, to say definitely that it was peritonitis. I am probably wrong, but I do not see any other way out.

As the cause of peritonitis the probability even at her age would be in favor of appendicitis. Also, since her symptoms were upper abdominal, in the examination of the abdomen it would be likely that the attention was paid during the cursory examination to the upper abdomen and that tenderness somewhere in the lower abdomen might have been overlooked. I think I must ascribe the symptoms to appendicitis in the absence of any earlier past history. I think it is peritonitis probably originating in a ruptured appendix.

Clinical Discussion

Dr. Tracy B. Mallory: We have here an obviously very blind case in which many guesses would be possible. I wonder if anyone else would like to make a diagnosis.

Dr. E. Parker: I think it quite possible she had carcinoma of the stomach with perforation. This complication occurs now and then, and would explain the peritonitis and also the blood in the vomitus.

Dr. George A. Leland, Jr.: It appears from the record that on the morning after her admission I saw this patient in consultation and stated that I was unable to make a diagnosis but that I felt very certain that in her condition at that time exploratory laparotomy would be of no avail. I vaguely remember the case. At that moment she was perfectly pulseless, with cold extremities. Effort was being made to build her up with some fluids. That was less than six hours before she died.

In retrospect, and considering the history as given today, I am inclined to agree with Dr. Cope that the hypertension and the diabetic condition are merely an unfortunate background of the individual and merely contributive rather than responsible for the very rapid dissolution that she underwent. As to what the acute condition was I am still very much in a quandary. With a patient of fifty-eight one thinks very seriously of malignancy of course. This woman had a red cell count of 5,000,000 and a hemoglobin of 75 per cent, which is fairly high for a malignancy of any long standing. On the other hand this blood picture is consistent with a cancer of non-ulcerated type without bleeding. The hematemesis is I think of course very significant in this case. I do not think we can pass it over lightly. I believe that the patient had a malignancy which involved her stomach, either primarily as cancer of the stomach with acute massive hemorrhage, or else an extrinsic cancer perhaps originating from the large bowel or the adjacent pancreas which ulcerated through into the stomach. I believe that the primary cause of death was an acute emergency due to a cancer.

Dr. William B. Breed: One might guess pancreatitis as tying two of the conditions together, namely the appearance of peritonitis and the disturbance in the sugar metabolism. I am not sure that the hematemesis could be explained on the basis of pancreatitis, but one does see occasionally hematemesis with pancreatitis.

Dr. Mallory: We certainly have seen cases of pancreatitis in which there has been a considerable elevation in blood sugar and the sudden appearance of sugar in the urine.

Dr. J. H. Means: I think that with the coffee grounds vomitus the chances of cancer are very great.

Clinical Diagnoses

Peritonitis, cause unknown.
Intestinal obstruction.
Diabetes mellitus.

Anatomic Diagnoses

Thrombosis of the superior mesenteric, portal, splenic and pancreatic veins.
Gangrene of the intestines.
Generalized peritonitis.
Ulcer of the esophagus with perforation into the posterior mediastinum and both pleural cavities.
Partial obstruction of the inferior vena cava by a sclerosed thrombus with collateral circulation through the left ovarian vein.
Chronic fibrous pleuritis.
Arteriosclerosis, marked aortic, slight coronary and renal.

Pathologic Discussion

Dr. Mallory: After having seen the autopsy on this case one can go back to one point in the story which I think one otherwise would overlook entirely, and that was that her presenting symptom had been pain between the two shoulder blades. She had two lesions which I am unable to connect directly. One, which I think probably explains that pain in the back and also explains the coffee grounds vomitus, was an ulcer of the esophagus which perforated into both pleural cavities. There was about 700 cubic centimeters of coffee grounds stomach material in the right pleural cavity and about 400 cubic centimeters of the same material in the left pleural cavity. The communication between the two pleural cavities was evidently digested by the gastric juice. That however

was not the cause of her death. In addition to that she had developed an acute mesenteric thrombosis starting in the superior mesenteric vein, running up the portal vein and involving the splenic vein. The small intestine from about one foot beyond the duodenum down nearly to the terminal ileum was completely gangrenous, and secondary to that arose the peritonitis which had been suspected. A further finding—probably a historical landmark only—was an old thrombotic occlusion of the inferior vena cava, with a well-developed collateral circulation through a greatly dilated left ovarian vein.

A PHYSICIAN: Did that ulcer of the esophagus show any gastric tissue?

DR. MALLORY: The postmortem digestion was so great that we were unable to recognize anything in its wall. I am quite sure it was not neoplastic. I think enough tissue was present for it to have been recognized if it had been.

DR. FULLER ALBRIGHT: Did she have a high temperature before she died?

DR. MALLORY: There was a terminal temperature of 105°.

DR. ALBRIGHT: I saw a case in Vienna something like this one. Professor Erdheim explained it in the following way. If a person for several days before death is extremely sick so that tissue resistance is decreased and if he at the same time has a high temperature, the gastric juice may run back into the esophagus and digest an opening through this organ. The gastric juice then flows down into the pleural cavity. The condition is most often seen in cerebral cases because of their tendency to develop hyperpyrexia.

DR. MALLORY: Although in this case we have a history of pain high in the back, suggesting that this is an earlier lesion.

DR. EDWARD L. YOUNG, JR.: We see the same thing in cases of lateral sinus thrombosis with tremendous elevation in temperature in the last few hours.

CASE 20202

PRESENTATION OF CASE

First admission. The patient, a sixty-six year old Canadian laundress, had been in good health until one year before admission. At that time she began to have a severe diarrhea. The discharge consisted of mucus, blood and thin watery feces. At times she was almost incontinent. This diarrhea continued, and was often associated with a sensation of fullness in the rectum. She had no other symptoms, and had been working until two weeks before admission. There had been no jaundice, weakness, abdominal pain or night sweats. She had lost twenty pounds in the past three months.

Twenty-seven years before admission she had pleurisy which necessitated a three months' stay at a hospital. She was discharged feeling well, but every winter since then had developed a cough associated with white sputum, occasionally blood streaked. During that year she fractured several ribs in an accident. For the past five years she had had dyspnea on climbing stairs. She had no edema. Three years before admission both knees became painful and swollen. A physician made a diagnosis of rheumatism. Two years before admission she fell down stairs and refractured two ribs. There was no history of pneumonia, chorea, or association with tuberculosis.

Physical examination showed the left pupil to be greater than the right. It did not react to light and was slightly irregular. The chest was slightly barrel shaped. The heart was enlarged to the left. There was a precordial systolic murmur, loudest at the aortic area. The aortic second sound was booming and greater than the pulmonic second. The blood pressure was 190/90. The abdomen was soft and slightly tender in the lower portion. No masses were felt. Rectal examination showed several large external hemorrhoids. Eight centimeters above the anus was a cauliflower polypoid mass which occupied the right half of the rectal wall. It was about 5 centimeters in diameter, bled easily, but did not completely obstruct the lumen. The knee jerks and ankle jerks were absent.

Examination of the urine was negative. The blood showed a red cell count of 4,500,000, a hemoglobin of 90 per cent and a white cell count of 5,500. A Hinton test was negative. The non-protein nitrogen of the blood was 25 milligrams. A phenolsulphonephthalein test showed 60 per cent excretion at the end of two hours.

An electrocardiogram showed normal rhythm, rate 70, and left axis deviation. A lumbar puncture was negative.

X-ray examination of the chest showed enlargement of the heart. The transverse diameter was 13.8 centimeters and that of the chest 23.8 centimeters.

Six days after admission a first stage abdominal perineal excision of carcinoma of the rectum was performed. The postoperative course was stormy. She developed pulmonary collapse and some pneumonia. On the seventeenth day after operation a second stage resection of carcinoma of the rectum was performed. Following this she was given a transfusion of 400 cubic centimeters of whole blood. This time she did well postoperatively. On the thirty-third day after the first operation she was discharged to a convalescent home.

Second admission, two months after her previous discharge.

She entered this time because of a fecal fistula in the posterior wound of the old abdominal perineal resection. The colostomy had nearly fallen back into the abdomen. The opening was very small. A plastic to the colostomy was performed under local anesthesia. She was dis-

charged improved two weeks after admission. The colostomy was working well.

History of interval. Three days after her discharge she began to have upper abdominal cramps which continued every few minutes through the whole day and evening. Soon after the onset of the pain she vomited. She vomited several times after this, at first food, then a small amount of blood and blood clot, and finally material which was fecal in character. Her colostomy was discharging feces at the time of entry.

Third admission, four days after her discharge.

Physical examination was similar to that at the second entry except that the heart impulse was felt 12 centimeters to the left of the midsternal line in the fifth interspace. The pulse was rapid, 110. The blood pressure was 125/100. The abdomen was moderately distended, with active peristalsis. It was soft and not spastic.

Examination of the blood showed a white cell count of 12,000. The non-protein nitrogen was 52 milligrams. The carbon dioxide combining power was 33.9 volumes per cent. The serum protein was 3.9 per cent.

On the day of admission an extraperitonealization of the cecum was performed under local anesthesia. The dilated cecum was sewed to the peritoneal surface and a right lateral lower quadrant wound packed open. Three hours later a tube was sewed into the cecum and placed on six centimeters water suction. Over fifty ounces of fluid feces was evacuated. The cecostomy failed to drain for the next thirty-six hours. There was increasing abdominal distention. The temperature and pulse rose to 104° and 140 respectively. Two days after admission the patient died.

X-ray Interpretation

Dr. George W. Holmes: These films were taken four months before death and may have no bearing on the immediate trouble.

The films taken of the abdomen show shadows which could be either in the kidney or the gall bladder, but which have the characteristic appearance of gall stones. We can be fairly certain that she had gall stones from this film alone.

In the film of her chest, also taken four months before death, the most striking thing is the marked increase in the supracardiac shadow, probably due to tortuosity of the aorta and not to actual dilatation. Here we have a very good chance to measure the diameter of the aorta between the esophagus and the outer side of the shoulder. I think that is slightly increased, but not more than one would expect at her age. The increase is not great. The curve of the left ventricle is a little prominent. One would interpret these findings as being due to arteriosclerosis with some hypertension, with possibly some dilatation of the aorta.

The position that the esophagus takes is also interesting. It seems to lie very close to the descending aorta. We have seen a few cases of this type, and we have been wondering whether there were adhesions or some abnormality of the mediastinum that fixed the esophagus to the aorta.

This is the oblique view and shows the aorta very well. This is the lower end of the esophagus in its normal position.

Differential Diagnosis

Dr. E. Parker Hayden: This history is of course a very typical one of cancer of the rectum, confirmed by operation, so that the obscurity of diagnosis dates from the postoperative complications.

This woman was sixty-six years old, and had, I judge, a certain amount of cardiovascular disease, which probably had very little to do with her subsequent death. In view of Dr. Holmes' x-ray interpretation, however, it may have had more to do with it than I think it did.

She had a two-stage operation of some type, probably the type which Dr. D. F. Jones originated and which is ordinarily done in this hospital, in which a loop colostomy is done at the first stage with a pelvic dissection and the rectum is taken out later, leaving a blind loop of bowel which extends from the colostomy down to the upper part of the pelvis. The colostomy subsequently retracted. That is a pretty common thing, particularly in persons with a fat abdominal wall associated with a short mesentery and producing retraction of the posterior wall with a tendency for the colostomy stoma to contract. This may have resulted in a mild amount of intestinal obstruction prior to the plastic operation.

The plastic, I assume, was a simple novocain procedure, done without entering the peritoneal cavity. It gave the patient a chance to move her bowels and probably made her condition temporarily satisfactory.

She entered the hospital four days after discharge with typical symptoms, it seems to me, of intestinal obstruction. Her abdomen showed no spasticity but was moderately distended. Her white cell count was moderately elevated, to twelve thousand. Her temperature was up very slightly. I should say that the picture was fairly typical of intestinal obstruction.

An operation was done under novocain anesthesia, presumably because she was very sick and because the obstruction had been going on for twenty-four hours. The cecum was apparently sewed to the peritoneal wall bordering the incision. We must assume that the cecum was definitely distended and that the operator thought the obstruction to be somewhere below the cecum. Apparently he thought that by anchoring the cecum and allowing it to seal before he opened it he would run less chance of con-

taminating the peritoneal cavity. That procedure, in view of the twenty-four hours of obstructive symptoms, is somewhat questionable. I think it would have been distinctly better to drain the bowel at that time, particularly in view of the evacuation of some fifty ounces of fluid a few hours later. There would be no more risk in putting a tube in at operation. That is the procedure that is usually carried out.

After the evacuation of fifty ounces of fluid the drainage stopped, the patient began to get worse, and then a period of thirty-six hours elapsed during which there was gradually progressive distention and no drainage. On the face of it I should say that there was probably an obstructing point somewhere in the small bowel above. It may have been that the patient was developing peritonitis without any particular evidence of spasm, but it seems to me more likely that there was intestinal obstruction higher up. Knowing the type of operation that was probably done, a very likely situation is that the small intestine may have rotated from right to left around the colostomy and formed a condition which although it might not obstruct all the time, at the same time was a definite factor in the obstruction. That sort of thing occurs with reasonable frequency in what essentially amounts to an intra-abdominal hernia around the left side of the colostomy with interference in circulation due to the drag on the mesentery. This may have been the situation. I should think from the history that it would probably have been wise to have gone in rather promptly after the drainage stopped and the distention increased, and that under novocain anesthesia one could have put a catheter in a loop of distended ileum somewhere.

Dr. Robert R. Linton: I saw this patient when she came into the Emergency Ward the last time. The picture at that time was certainly one of intestinal obstruction, most probably of the small bowel, and that was why operation was advised. Dr. Stewart, who did the operation, reported that the small bowel was somewhat dilated, but not so much as the cecum. The cecum was so tense and dilated that it was impossible to get a tube into it, so he thought it was much safer to suture it to the peritoneal surface and open it later. The colostomy was irrigated before operation, with practically no return. It seemed pretty definite that she had mechanical obstruction.

A Physician: Would the dilated cecum more or less rule out a point of obstruction above it?

Dr. Hayden: It would, unless a small intestinal obstruction occurred a little later, when the free flow from the cecostomy stopped. There was no reason why any quantity of material would collect in the cecum unless there was obstruction below it.

Clinical Diagnoses

Acute intestinal obstruction.
Carcinoma of the rectum.

Anatomic Diagnoses

Mesenteric thrombosis (embolism?)
Operative wounds: old resection of anus, rectum and sigmoid (cancer of rectum); recent cecostomy.
Peritonitis, slight generalized.
Arteriosclerosis, marked aortic, moderate coronary, mesenteric and renal.
Cardiac infarct, old.
Mural thrombi, left ventricle and aorta.
Healed pulmonary tuberculosis, left.
Chronic fibrous pleuritis, bilateral.
Cervical polyps.

Pathologic Discussion

Dr. Tracy B. Mallory: The obstruction, if one is to call it obstruction, in this case was similar to that in the last. It consisted in an infarction of the entire intestinal tract, in this case from the duodenum not merely down to the terminal ileum but also including the major part of the large intestine. Whereas in the last case the infarction was of venous origin from a thrombosis to the branches of the portal vein, in this case it was of arterial origin from occlusion of the mesenteric artery. The occlusion may have been a local thrombotic affair, since a fairly marked degree of arteriosclerosis was present in the mesenteric artery itself and numerous atheromatous ulcerations were formed in the aorta. Or it is possible that it may have originated in an embolus, for which she had a perfectly good focus in an infarct of the heart with an overlying mural thrombus. From this a clot might well have slipped off and become the starting point for the gradually progressive thrombosis of the mesenteric artery. The coronary arteries were free from occlusion. This infarct was a very old one. I think we must assume that she had at one time a coronary occlusion followed by almost complete recanalization of the artery.

A Surgeon: There were no metastases?

Dr. Mallory: No.

Dr. Holmes: Was there anything to account for the unusual position of the esophagus?

Dr. Mallory: We did not notice anything.

The third case of this group, in which much of the general discussion of the group appears, will be published next week.

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The Massachusetts Medical Society

DISTINGUISHED SPEAKERS FOR THE ANNUAL MEETING

THE Annual Dinner of the Massachusetts Medical Society will take place on June 5, 1934 at 7 P. M., in the Ballroom of the Hotel Bancroft, Worcester. It is the chief social function of the Society. In recent years, however, the attendance has been disappointingly small; possibly because the charge has been high for these times, possibly because the names of the speakers have not been made public.

This year the tickets have been reduced to two dollars, and an excellent dinner will be served.

The President of the Society has announced that the following distinguished speakers will be his guests:

His Excellency The Governor of Massachusetts
His Honor The Mayor of Worcester
The Honorable James M. Morton, Jr., Judge of the United States Court of Appeals
The Right Reverend Henry K. Sherrill, D.D., Episcopal Bishop of Massachusetts
Dr. Lewis Perry, Headmaster of Phillips Exeter Academy

It is also to be remembered that the Shattuck Lecture on Monday evening will be given by Dr. Warfield T. Longcope, Physician-in-Chief, The Johns Hopkins Hospital and Professor of Medicine of Johns Hopkins University School of Medicine. His subject will be "The Importance of Disturbances in Nutrition in Oedematous States."

Dr. Lincoln Davis, President of the Boston Medical Library and Consulting Surgeon, Massachusetts General Hospital, will present the Annual Discourse on Wednesday at 1 P.M. His subject will be "The Objectives of Medical Progress."

With this impressive list of speakers, a large registration of the Fellows of the Society is reasonably to be expected.

THE DANGER OF DINITROPHENOL AS A REDUCING AGENT

THE World War through the extensive manufacture of trinitrotoluol and related products served to call attention to the marked toxic character of these substances. Their absorption was characterized by extensive liver damage, pigmentation of the skin. An accompanying increase in pulse rate and temperature emphasized their general biological effect in enhancement of oxidative processes.

Dinitrophenol, a closely related compound to T.N.T., has long been known in the laboratory but only recently has been brought to popular attention through the articles by Cutting and Tainter[1, 2] in the *Journal of the American Medical Association.* These articles emphasized the value of this chemical as a metabolism raising agent through acceleration of oxidative processes. They brought forward strong evidence for its utility, raising the basal metabolic rate 30 to 50 per cent without symptoms. Under ordinary conditions fat is apparently oxidized without the development of acidosis.

Unfortunately strong emphasis was not laid on the toxicity of this compound although warning was given as to its danger. Already cases of death due to its use are being reported here and abroad[3].

This drug has great potential danger because of a narrow margin between the toxic dose and the therapeutic dose. Fortunately it is fairly rapidly excreted by the kidneys so the possibility of accumulative effects is relatively slight. In the cases of death thus far reported liver damage appears to be the outstanding factor

although severe renal damage, particularly in the form of tubular nephritis, is also an important factor.

The chief danger lies in the uncontrolled sale of this powerful agent to the laity. There is nothing that will prevent its use in the various patent medicines for reducing. There is nothing to prevent its over-the-counter sale by druggists. Potentially dangerous when prescribed by physicians to patients under strict supervision, it may be deadly to those taking it without competent advice. All too often the uninitiated and enthusiastic seeker of slimness falls into the same difficulty as that met with by the lay user reported by the London correspondent in the *Journal of the American Medical Association.* Although the dose was stated to be one capsule a day, she took seventeen capsules in a three-day period and died.

It is to be hoped that as wide publicity will be given to the dangers of this metabolism stimulating substance as has been given to its possible advantages.

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1 Cutting, W. C., Mehrtens, H. G., and Tainter, M. L.: J. A. M. A. 101:193, 1933.

2 Cutting, W. C., and Tainter, M. L.: J. A. M. A. 101:2099, 1933.

3 J. A. M. A. 102:1314, 1934.

THE TAX IMPOSED BY TUBERCULOSIS

THE article by Dr. C.-E. A. Winslow on page 1084 of this issue presents in impressive form the tax levied on communities by tuberculosis. Unlike the increasing taxation of the federal and state governments, Dr. Winslow shows that the burden of tuberculosis has been diminishing, and may be less if we continue to concentrate on the problems incident to this disease.

Until Koch's revelation of the pathology of the disease, little progress had been made in combating it. In the light of the discovery of the bacillus, physicians and public health authorities united in patient study of the possibilities of control, with the result that many thousands of lives have been saved and the great proportion of the tuberculosis tax has been lifted. Fortunately, Koch's discovery opened the fields of scientific research and led to other triumphs in dealing with disease which in turn diminish the burden of morbidity and mortality. It is well that due credit should be given to scientific medicine through its assistance in diminishing the taxable burden of disease.

Further progress will be made if and when more general knowledge permeates society at large followed by more generous coöperation with physicians and public health agencies.

TRIBUTE TO DR. WILLIAM HENRY WELCH

SUPPLEMENTING the editorial of last week and the letter by Dr. Joseph H. Pratt, we are privileged to present three more references to Dr. Welch which appear on pages 1087 to 1089.

Dr. Welch visited Boston on many occasions and delivered several addresses here. His Ether Day Address at the Massachusetts General Hospital in 1908 and that on the importance of medical libraries in 1930 will be remembered by those privileged to hear them.

Those who were associated with or studied under him are unanimous in attributing to this eminent man great influence in the progress of medicine in this country.

His love for Johns Hopkins University School of Medicine is shown by the bequest of one-fourth of his estate, including his medical, scientific, and literary books, and medals to this organization.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

BOWLER, JOHN P. A.B., M.Sc., M.D. Harvard University Medical School 1919. F.A.C.S. Dean, Dartmouth Medical School. Surgeon, Hitchcock Hospital, Hanover, New Hampshire. Address: Hitchcock Clinic, Hanover, New Hampshire. Associated with him is GILE, JOHN F. A.B., M.D. Harvard University Medical School 1920. F.A.C.S. Instructor, Dartmouth Medical School. Surgeon, Hitchcock Hospital, Hanover, New Hampshire. Address: Hitchcock Clinic, Hanover, New Hampshire. Their subject is "Compression Fractures of Vertebral Bodies." Page 1052.

MORSE, JOHN L. A.M., M.D. Harvard University Medical School 1891. F.A.A.P. Professor of Pediatrics Emeritus, Harvard Medical School. Consulting Physician Children's Hospital, Infants' Hospital and Beth Israel Hospital. Member, New England Pediatric Society, American Pediatric Society, Association of American Physicians and Boston Obstetrical Society. His subject is "The Necessary Requirements in a Minimum Diet for Infants and Children." Page 1057. Address: 319 Longwood Avenue, Boston, Massachusetts.

LENNOX, WILLIAM G. A.B., A.M., Sc.D., M.D. Harvard University Medical School 1913. Instructor in Neurology, Harvard Medical School. Junior Visiting Neurologist, Boston City Hospital. His subject is "The Use of Ergotamine Tartrate in Migraine." Page 1061. Address: 909 Medical Building, Boston City Hospital, Boston, Massachusetts.

JOHNSON, ALLEN S. A.B., B.A., M.D. Harvard University Medical School 1927. Assistant Visiting Physician, Springfield Hospital. Visiting Physician, Tuberculosis Division, Springfield Isolation Hospital. His subject is "The Parenteral Administration of Paraldehyde for the Control of Pain and Convulsive States." Page 1065. Address: 121 Chestnut Street, Springfield, Massachusetts.

HALL, WILLIAM E. A.B., M.D. Yale University School of Medicine 1925. Yale Cardiologist, Meriden Hospital. Attending Physician, Meriden Hospital. Address: Professional Building, Meriden, Connecticut. Associated with him is

MURDOCK, THOMAS P. M.D. University of Maryland School of Medicine 1910. Chief of Medical Service, Meriden Hospital, Meriden, Connecticut. Address: Meriden Hospital, Meriden, Connecticut. Their subject is "Foreign Protein Sensitization with Meningeal Involvement Due to the Use of Vaccine." Page 1067.

COTTON, FREDERIC JAY, M.D., and MORRISON, GORDON MACKAY, M.D. See page 817, issue of April 12, for records of authors. Their subject is "Recurrent Dislocation of the Shoulder." Page 1070.

CHEEVER, AUSTIN W. A.B., M.D. Harvard University Medical School 1914. Assistant in Dermatology and Syphilology, Harvard University. Assistant Dermatologist, Children's Hospital. Visiting Dermatologist, Beth Israel Hospital. Consulting Dermatologist, Framingham-Union, Waltham, Goddard, and Brockton Hospitals. His subject is "Progress in the Diagnosis and Treatment of Syphilis, 1933." Page 1072. Address: 472 Commonwealth Avenue, Boston, Massachusetts.

MISCELLANY

ONE WAY TO REDUCE TAXES

BY DR. C.-E. A. WINSLOW*

The first definition of a "tax" is a "contribution levied on persons, property, or business for support of government." A second meaning of the word is a "strain or heavy demand" upon a person or a community. We hear a great deal today about the first kind of taxes. It might be wise to give some thought to the second.

From the standpoint of "strain or heavy demand" some of our most exacting tax collectors are the germs of the communicable diseases. In 1900 the bacillus of tuberculosis taxed the people in the United States to the tune of 195 lives for every hundred thousand persons in the population. That is this tax collector took the life of one person out of every 500 each year. In addition it collected the cost of medical and hospital care and the cost of supporting in partial or complete idleness at least one person out of every 150.

By 1930 the death rate from tuberculosis† had fallen from 195 to 67 per 100,000. We had reduced the tax paid to this public enemy to one life out of every 1500 persons in the population with a similar reduction of two-thirds in the burden of sickness and disability. This is a sort of tax reduction which is well worth while.

Courtesy Henry Street Visiting Nurse Service

Public health is not just a vague name to people who are familiar with the Public Health nurse.

How has this result been accomplished? By legislative machinery for the reporting of cases, by segregating or educating the consumptive so as to avoid infection of others, by eradication of bovine tuberculosis and pasteurization of milk, by public health nursing service to seek out contacts and early cases and bring them under medical care, by clinic service for early diagnosis, by sanatoria for treatment, and by systematic follow-up and supervision of the arrested case to hold the disease process under control. Simple, efficient, well-tried public health machinery has accomplished the task. Its efficacy has been fully tested during the past three years when the potential effects of the depression upon the death rate have been warded off except in certain relatively small sections of the population where deprivation has been greatest. It is neither chance nor some hypothetical biological change which has brought about results; for these results, in individual communities, have again and again been shown to vary in relation to the provision of community machinery for controlling the disease.

Recently, a new weapon has been placed in our hands. Experience has shown that many of the deaths which still occur from tuberculosis are due

*Professor of Public Health, School of Medicine, Yale University.

†Based on figures for Registration States of 1900.

to previous partially controlled infection which suddenly lights up in a form too severe to be checked by our routine methods. The hope of preventing such catastrophes lies in the earlier detection of incipient infection—preferably in childhood, when the seeds of later trouble are generally sown. The new methods of taking x-ray pictures of large groups of school children at a minimum cost brings promise of a better defense against the tubercle bacillus than we have ever been able to set up before.

The cost of a well-organized tuberculosis program (excluding the cost of sanatorium care which is largely treatment rather than prevention) is less than 10 cents per year for each person in the general population. The results far outweigh this insignificant expenditure. We may illustrate by a single instance. The reduction in the death rate from pulmonary tuberculosis in Syracuse, N. Y., for the decade 1922-1931 as compared with the decade 1912-1921 amounted to a saving of 115 lives a year. This takes no account of the far greater savings accomplished between 1892 and 1912 but merely measures the additional accomplishment after a more intensive community program was inaugurated in 1923. On a conservative estimate, based on tables of the value of a human life at different ages, this additional reduction amounted to an economic saving of over a million and a half dollars a year to the community—based on life-saving alone and with no allowance for the burdens of disability and medical care. The total cost of the enlarged and almost ideal tuberculosis-control machinery of Syracuse was less than twelve thousand dollars. By paying one dollar in taxes to the city and the Community Chest, Syracuse saved paying one hundred dollars in taxes to the tubercle bacillus.

No matter how much the budget is cut, children must not suffer. This little boy is being protected against tuberculosis by having a tuberculin test.

The question then is not whether we shall pay taxes or not, but how much we shall pay and for what. We can contribute a small sum to our health departments and clinics and nursing associations and tuberculosis associations for prevention; or we can pay a much larger sum as a tax on unprevented disease for the care of the sick, the support of the invalid, the burial of the dead, and the loss of man power. We have reduced the tax levied by disease during the past thirty years to a fraction of what it once was. We cannot afford to let our progress be nullified by the panic parsimony which is the enemy of true economy. We must rather go on with all the weapons at our command to the ultimate conquest of tuberculosis. "A few cents for defense, rather than millions for tribute," might well be our motto. The tax laid upon human vitality by preventable disease is one which we can abate if we have the courage to support in this crisis the official and the voluntary health agencies which have defended us so successfully in the past.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DIVISION OF ADULT HYGIENE

NUMBER 13 *Cancer Clinic Bulletin*, MAY 1, 1934

CANCER CLINICS AT BOSTON AND PONDVILLE

On Wednesday, April 11, 1934, the last of the three Cancer Clinics, arranged for the season of 1933-1934 by the Cancer Committee of the Massachusetts Medical Society, was held at the Massachusetts General Hospital in Boston, and at the State Cancer Hospital in Pondville. About forty-five of the physicians on the staffs of the organized Cancer Clinics in Massachusetts were in attendance.

The Clinic at the Massachusetts General Hospital began at 9:30 A.M., in the Surgical Amphitheatre, with an operative clinic. Dr. E. M. Daland operated for cancer of the lip with radical neck dissection.

Dr. Channing C. Simmons did a radical amputation for cancer of the breast; and Dr. J. V. Meigs demonstrated the application of radium in the treatment of a case of cancer of the cervix of the uterus.

Demonstrations were then given in the Surgical Amphitheatre as follows: Dr. E. P. Hayden showed an unusual tumor of the mouth, which was removed beneath the angle of the jaw, and proved to be probably a fetal adenoma of the aberrant thyroid. He also demonstrated the specimen of a branchial cyst which he had recently removed by operation.

Dr. E. M. Daland demonstrated a plastic operation for cancer of the lip, and showed a case of extensive lymphangioma.

Dr. Grantley W. Taylor showed a case of disease of the ulna due to syphilis in which the differential diagnosis of bone sarcoma was suggested by the x-ray plates; also an advanced and inoperable case of cancer of the breast in a young woman who was given x-ray treatment to produce an artificial menopause. He also demonstrated a case of advanced cancer of the tonsil, which had had (1) preliminary x-ray radiation, (2) a dental cleansing of the mouth, followed by (3) a two-thousand r-unit treatment with x-ray, and (4) the insertion of radium seeds. This treatment produced a severe reaction followed by marked symptomatic improvement. He also demonstrated another case of inoperable cancer of the

pharynx and palate of Grade II Malignancy in a man, which also improved under the plan of treatment used in the previous case. Dr. Taylor also spoke of the neurosurgical relief of pain by alcoholic injection or resection of the branches of the fifth nerve. The Clinic then was adjourned to Pondville, where a luncheon was provided by the courtesy of the State Department of Public Health.

The following short papers were given:

Dr. Grantley W. Taylor—Cancer of the Mouth in Women.

Dr. Roger Graves—Treatment of Cancer of the Penis. (Two cases shown.)

Dr. Joe V. Meigs—Carcinoma of the Cervical Stump.

Dr. Ernest M. Daland—Lymphatic Drainage in Cancer.

Drs. Richard Dresser and Charles Dumas showed three cases that were receiving x-ray treatment for cancer of the upper esophagus, cancer of the buccal mucosa and cancer of the tongue.

Dr. Daland showed two cases of extensive cancer of the lower lip, one before and one after operation; a rope graft under construction for a deformity of the buccal mucosa, and an extensive carcinoma treated by x-ray, surgery, and a plastic operation.

Dr. Taylor showed an inflammatory type of cancer of the breast and another breast case with radium needles in situ.

Dr. Meigs showed a patient six months pregnant with an early cancer of the cervix and invited discussion as to the proper treatment.

Dr. Graves showed a cancer of the prostate after perineal prostatectomy.

Dr. Merrill demonstrated a lymphoma case and a possible cancer of the lung.

The residents showed a melanotic sarcoma of the thigh, a cancer of the jejunum (postoperative).

The meeting adjourned at 5:00 P.M.

On Tuesday, April 17, by invitation of the Lawrence Cancer Committee, seventeen members of the Essex North District attended the cancer clinic where Doctor Channing Simmons acted as consultant.

Multiple Cancers

The study of multiple primary malignant tumors has gradually shifted from the mere accumulation of case records to a comprehensive study of the implications of this somewhat unusual occurrence. Interest is no longer so much aroused by the mere occurrence of multiple cancers. The significance of the frequency or infrequency of multiple tumors now holds the center of the stage, as occurrence of these tumors may throw light on the but vaguely understood problems of resistance or susceptibility to cancer. Several comprehensive studies of the subject have been made, one of the first being that of Major[1], followed by Owen[2], and the most recent by Warren and Gates[3]. In addition to these a special study of multiple cancers of the mouth, based on material from the Huntington Hospital, has been made by Lund[4], whose findings are in essential accord with those of Warren and Gates.

In the paper by the latter authors, 1259 cases of multiple malignant tumors are collected from the literature, together with their own cases.

Studies of the frequency of this condition are even more relatively unsatisfactory, as various authors differ considerably in the criteria that they lay down for the definition of this condition. The most rigid criteria are those of Billroth, that each tumor must have a different histologic appearance, that the tumors must arise in different locations, and that each must produce its own metastasis. This last requirement particularly rules out a very large number of cancers and results in a falsely low recording of the incidence of multiple malignant tumors. A perhaps fair standard is that set up by Warren and Gates, namely, each of the tumors must present definite evidence of malignancy, each must be distinct, and the probability of one being a metastasis of the other must be excluded.

Some patients show an extraordinarily large number of different cancers, the most numerous being a case reported by Rappin[5], who showed over 200 separate carcinomata.

Up to 1932, 111 cases of three or more multiple malignant tumors had been reported. Double cancer is, of course, much more frequent than this first group. Among the double cancers, multiple cancers of the gastrointestinal tract are particularly frequent. This is perhaps caused by the frequent origin of carcinoma from mucosal polypi of the intestine. Warren states he has seen not infrequently multiple cancers of the large bowel associated with multiple polypi.

In the different series of cases reported in sufficient detail to estimate the frequency of this condition the variation is strikingly wide. Some authors find the multiple cancers to run less than one per cent of all malignant cases, while others find as high as over four per cent. In general, with due regard to the material utilized, about three per cent of all cases of cancer show more than one malignant tumor.

Whether this percentage is higher or lower than that of chance is, of course, of real importance as indicating whether the person with one tumor is more than normally likely to have a second, that is, unusually susceptible to the disease, or is less than normally likely to develop a second cancer, that is, abnormally resistant to the disease.

To calculate accurately the occurrence of multiple cancer on the basis of chance alone is extraordinarily difficult, yet this must be done to establish a norm. Such a norm has been established, though not with complete accuracy. Multiple cancers are found on the basis of this calculated norm to occur about three times as frequently as they would be expected to on the basis of chance alone, allowing for a large number of corrections. It may be definitely concluded, therefore, that there is some predisposition

or susceptibility to cancer in certain persons, or else that the action of some factor favoring the development of malignancy is of importance.

The multiple cancers occur at practically the same age as do single cancers, and the average duration from the onset of the earlier tumor to death is about three years.

The lesson to be drawn from these findings is that even though a person has been cured of cancer he is not free from danger of that disease; in fact he is three times more likely to develop a new cancer than a person who has never had cancer.

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2 Owen, L. J.: J. A. M. A. 76:1329, 1921.
3 Warren, Shields and Gates, Olive: Am. J. Cancer. 16:1358, 1932.
4 Lund, Charles C.: New Eng. J. Med. 209:1144, 1933.
5 Rappin: Gaz. Méd. de Nantes, Aug. 15, 1928. Abstr. by Cancer Review. 4:324, 1929.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1933 AND SEVEN YEAR AVERAGE

MONTH ENDING APRIL 28, 1934

Diseases	1934 Week ending Apr. 7	1934 Week ending Apr. 14	1934 Week ending Apr. 21	1934 Week ending Apr. 28	Average cases reported for week corresponding to Apr. 28 for past seven years	1933 Week ending Apr. 8	1933 Week ending Apr. 15	1933 Week ending Apr. 22	1933 Week ending Apr. 29
Actinomycosis	1	—	—	—	—	—	—	—	—
Cerebrospinal Men.	1	2	1	1	1	—	1	—	—
Chicken Pox	71	88	91	127	94	159	136	190	158
Conjunctivitis Inf.	8	—	—	3	5	—	—	1	—
Diphtheria	3	3	1	3	16	4	5	5	7
Dysentery Bacillary	—	—	—	1	—	—	—	—	—
Encephalitis Epid.	—	—	1	—	—	—	—	—	1
German Measles	1	4	2	—	32	9	4	7	6
Influenza	—	6	2	—	7	19	7	10	5
Measles	23	55	52	71	292	275	242	266	273
Mumps	118	144	126	131	81	117	88	86	84
Paratyphoid Fever	—	—	—	—	—	—	—	2	—
Pneumonia (Broncho)	19	30	24	17	31	19	22	22	24
Pneumonia (Lobar)	29	31	45	26	41	40	25	32	20
Scarlet Fever	77	64	91	58	88	167	140	118	134
Septic Sore Throat	1	2	1	—	2	2	—	5	1
Smallpox	—	—	—	—	1	—	—	2	9
Tetanus	—	—	—	1	—	—	—	—	1
Trichinosis	—	—	—	—	—	—	1	—	—
Tuberculosis (Pul.)	28	23	24	16	33	34	15	37	30
Tuberculosis (O. F.)	4	1	2	1	4	1	2	2	3
Typhoid Fever	2	1	1	—	1	—	—	1	—
Undulant Fever	1	—	3	—	—	1	—	—	1
Whooping Cough	95	44	66	81	69	112	88	81	112
Gonorrhea	25	13	21	34	30	23	16	33	23
Syphilis	56	42	48	75	45	43	23	32	62

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

CORRESPONDENCE

TRIBUTES TO THE MEMORY OF DR. WILLIAM HENRY WELCH

May 7, 1934.

Editor, *New England Journal of Medicine*,

There are so many things to be said of Dr. Welch that one must exercise great restraint not to talk about him too much. To the student who knew him only as a lecturer, perhaps the marvelous amount of information he possessed was most impressive; better acquaintance revealed the remarkably clear mind, wide outlook and sound judgment. A quiz by Dr. Welch was always welcomed by the students because they were sure he would be not only charitable toward their ignorance but most helpful in suggesting new points of view, new lines of investigation, and hitherto unknown sources of information.

His positive and considerable contributions to sci-

entific knowledge were made in the earlier years of his activity, but it was in the field of the intangible that he was most powerful. He was a great university teacher, not in the Roman sense of "discipline," which, however, he appreciated but in the Athenian sense of "inspiration," possessing a most vigorous and stimulating mind. He was an important member of that small group who placed the study of medicine on a university basis in this country. He said that the secret of keeping young was to acquire a new technic. In any case, he remained young and was successively Professor of Pathology, Director of the School of Hygiene and Public Health, and Professor of the History of Medicine.

To those who had the privilege of the friendship of Dr. Welch he will always be the great humanist. Life became better because one had shared it, though in slight degree only, with him. One of the traditions is that he never spoke ill of any man, and his generous charity was but one of the manifestations of a rich and charming personality. He was in the great tradition, and his memory will be cherished always by his pupils with deep affection.

Yours truly,

STEPHEN RUSHMORE, M.D.

520 Commonwealth Avenue, Boston.

May 4, 1934.

Editor, *New England Journal of Medicine.*

In my search for information concerning medical schools, it was natural for me to turn to the family physician who had inspired me to study medicine. His statement was "try to get into the school where Dr. William H. Welch teaches, nothing else matters". This in a line shows the tremendous influence at that time of this great man throughout the country.

During my four years in the Johns Hopkins Medical School, Welch was a great power. At no time did any student lose an opportunity to listen to anything he had to say. Occasionally there would be a chance to see him do an autopsy, hear him give a lecture or a quiz. Often an informal discussion, not always medical but invariably instructive and pleasing. The fondest memories, however, are those of "Popsy's" little summaries of the important lectures and demonstrations. Usually the student was lost in a maze of technicalities and would have derived small benefit in many instances had not Dr. Welch told us in a few simple words, what it was all about.

He was adored by all of us and we are grateful for his long and useful life.

Yours truly,

ARTHUR W. ALLEN, M.D.,
Class of 1913.

264 Beacon Street, Boston.

WILLIAM HENRY WELCH

1915—Baltimore. The laboratory; Johns Hopkins Hospital; the weekly clinical pathological conference. Around the central table, "pipe stand" seats are crowded with clinical clerks, interns, assistants from all departments of the hospital. In chairs on the floor are department heads: — Halsted, Kelly, Howland, Thayer, Barker, Meyer and among them benign, alert, genial "Popsy" Welch; bright of eye, pointed of beard and rotund of abdomen.

The history of the patient is presented by the intern; a case of cirrhosis of the liver with jaundice. The description of the autopsy follows by the resident pathologist. Organs are demonstrated and microscopic slides shown on the screen. The case is discussed in general and then follows a summary by Dr. Welch in his judicial, crystal-clear words, interspersed with many remarks concerning the etiology of liver disease, the various recent important articles on the subject, quoting both writer and journal, and something of the history of the development of the most plausible explanation of jaundice.

Or again; it is a beautiful spring evening and the Pithotomy Club (a student society) is having its annual party on board the "Kitty Knight," a disreputable but long-lived and famous Chesapeake Bay steamboat. Students are everywhere, and also a goodly sprinkling of faculty, among them "Popsy." Supper consists mostly of "Wienies" and beer, dispensed by the ubiquitous Louis Hanselmann.

Soon everyone crowds forward to the cabin to get seats for the play. Dr. Welch has but recently returned from a trip to China and Japan, made with Dr. Flexner, in the interest of medical education in the East, and the title of the play this year is "Popsy in the Orient." The cast is wholly in Chinese costume and the antics of the two students who impersonate Dr. Welch and Dr. Flexner, so humorous and in spots so lacking in reserve that the show "brought down the house". At its finish Dr. Welch in his so characteristic, dry humor made a few congratulatory remarks, coyly disclaiming for his friend Dr. Flexner and for himself any such actions while away, as had been suggested by the skit.

1923—London. The marvelous Jacobean guild-hall of the Merchant Tailors on Threadneedle Street in the "City" is resplendent with lighted torches and dining tables round which are gathered about three hundred "old students" of St. Bartholomew's Hospital. It is their dinner held during the "Solemnities" which celebrate the eight hundredth anniversary of the founding of the hospital. At the high table alongside dignitaries of church, state, and medicine, sits "Popsy," representing the General Education Board and the American Universities. His dignified and very forceful speech pointing out the many bonds uniting America with England and emphasizing the fact that science and the search for truth has no boundaries, geographical or otherwise, was by far the best of the evening.

1933—A sunny afternoon in Baltimore. In a hospital bed sits "Popsy," smoking the beloved cigar, surrounded by books and flowers in every available space. He has grown a bit gaunt, for at last the

Grim Reaper has signalled his coming, but nevertheless there are still many things and people worth talking and reading about and many, many memories to recall and recount.

And so we leave him — among his friends and his flowers — (on his eighty-fourth birthday so many were sent that he ordered them distributed through all the wards of the hospital) and his marvelous memories; memories of a long life wholly devoted to service for his fellow man, and influential beyond that of all others.

ANONYMOUS.

FREE LUNG DEFLATION TREATMENT

The Central New England Sanatorium, Inc.,
and Industrial Colony
Rutland, Massachusetts

May 1, 1934.

Dear Mr. Editor:

We wish to call attention to the physicians of New England that we can accept their cases of early tuberculosis of the lung for deflation treatment without cost of any kind to the patient. We propose to keep these patients at the sanatorium for ten days or two weeks and then return them to the family physician for refills and further care. We sincerely hope that the family physician will present himself at the sanatorium where he will be given a demonstration of the technique of the operation and the technique of refills.

Application should be made to the Medical Director, Dr. Bayard T. Crane, Rutland, Mass. Tel. Rutland 124.

Truly yours,
WALTER C. BAILEY, M.D., *President*,
BAYARD T. CRANE, M.D., *Medical Director*.

OFFICIAL ACTION OF THE BOARD OF REGISTRATION IN MEDICINE

The Commonwealth of Massachusetts
Department of Civil Service and Registration
Board of Registration in Medicine
State House, Boston

May 2, 1934.

New England Journal of Medicine,

This is to inform you that at a meeting of the Board of Registration in Medicine, held April 26, 1934, the license of Dr. Percy Whitman Carr of Hyde Park, Boston, was revoked for conviction in court for administering medicine to produce a miscarriage.

Very truly yours,
STEPHEN RUSHMORE, M.D., *Secretary*.

MAY THE DOCTOR REMOVE THE QUARANTINE CARD?

To the Editor:

In the issue of May 3 of the *New England Journal of Medicine*, an "Anonymous" M.D. comments on a discussion that followed an address by Dr. Wilinsky on the Relation of Public Health to Private Practice. Says the "Anonymous":

"The discussion that followed was illuminating. One physician said that he should (for the price of a visit) be allowed to take down a measles quarantine card rather than have that service performed by a public health nurse. Does he not realize that it is because of that attitude that he is not employed more? If he were a layman would he feel that it was fair to have to pay for the visit of a supposedly skilled man to have that unskilled function performed?" . . . .

The writer of this letter was the first one to discuss the paper and he was the one who objected to the Board of Health's policy of having a quarantine card removed by a nurse without hearing first from the attending physician. There was no mention on my part of pecuniary considerations. It was the self-respect and the dignity of the profession I was after. I maintained that a physician is certainly more qualified to judge than a nurse, when his measles, scarlet fever, or any other reportable case is ready for discharge. The nurse's function was always considered to carry out the physician's orders, but never to make diagnoses for him.

It is not a matter of the physical removal of the quarantine card, it is the judgment as to when this removal should occur. I shall gladly grant the first one to the nurse, when the second is performed by the attending physician. Until recently such was always the policy of the Boston Board of Health. Why the change now?

M. J. KONIKOW, M.D.

Roxbury, May 6, 1934.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
April 30, 1934.

Managing Editor,
The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of March 31 the following have been accepted:

Cheplin Biological Laboratories, Inc.
- Ampule Solution Procaine Hydrochloride 2 Per Cent, 1 cc.
- Ampule Solution Procaine and Epinephrine, 3 cc.
- Ampules Bismuth Subsalicylate 2 Grains (0.13 Gm.) in Oil, 1 cc.
- Ampules Solution Mercury Succinimide 1/6 Grain (0.01 Gm.), 1 cc.

Lederle Laboratories
- Refined Diphtheria Toxoid (Alum Precipitated)

Schering & Glatz, Inc.
- Urotropin
  - Urotropin Tablets, 5 grains (0.3 Gm.)
  - Urotropin Tablets, 7½ grains (0.5 Gm.)

Ucoline Products Company
Ucoline Calcium Phosphate Cocoa Wafers
Yours very truly,
PAUL NICHOLAS LEECH, *Secretary*,
Council on Pharmacy and Chemistry.

RECENT DEATHS

CUSHING—HAYWARD WARREN CUSHING, M.D., of 70 Commonwealth Avenue, Boston, died at his home, May 9, 1934. He was born in Boston in 1854, the son of Hayward and Harriett (Pierce) Cushing. He was fitted for college at the Boston Latin School, and graduated from Harvard College in 1877 and the Harvard Medical School in 1882. He studied later in England, Germany, and Austria. After returning to Boston he served on the staffs of the Boston Dispensary, the Carney Hospital, and later as surgeon to out-patients, and visiting surgeon to the Boston City Hospital. After concluding active service at the last-named institution, he was given the title of consulting surgeon. He withdrew from active practice to a large extent in 1905 due to ill health.

He joined the Massachusetts Medical Society in 1881, and was a Fellow of the American Medical Association, a member of the Société Internationale de Chirurgie, the American College of Surgery, the Boston Surgical Society, the Boston Society for Medical Improvement, the American Society for the Advancement of Science, and the Boston Society of Medical Sciences. He held membership in the Harvard Club, the Tavern Club, and the Oakley Country Club.

He is survived by his widow Mrs. Martha (Thompson) Cushing, and two daughters, Mrs. Helen C. Bird of Norwell, and Mrs. Elizabeth C. Carleton of Cambridge.

SCRIBNER—JAMES MERLE SCRIBNER, M.D., of 11 Kilgore Avenue, West Medford, formerly of Lowell, Massachusetts, died at his home, May 11, 1934. He was born in 1902, the son of Mr. and Mrs. Charles E. T. Scribner. His early education was acquired in the public schools of North Adams and Medford.

He engaged in premedical subjects at Tufts College and later graduated in Medicine from Tufts College Medical School in 1927, where he was president of the Phi Chi Fraternity. His internship was at the Lowell Corporation Hospital, and he later served as resident physician and superintendent. After the name of the hospital was changed to St. Joseph's, he served as chief of the obstetrical service. He joined the Massachusetts Medical Society in 1927, and was a Fellow of the American Medical Association. He was a junior member of the American College of Surgeons, and was affiliated with the Masons and Elks. He is survived by his parents and two brothers, Dr. Edwin M. Scribner, and Charles E. Scribner.

SLATE—AMES WILSWORTH SLATE, M.D., of 74 Oak Street, Indian Orchard (Ludlow), died at his home, May 11, 1934. He graduated from the Harvard Medical School in 1900.

NOTICE

AMERICAN MEDICAL GOLFING ASSOCIATION

The American Medical Golfing Association will hold its twentieth annual tournament at the Mayfield Country Club in Cleveland on Monday, June 11, 1934.

Thirty-six holes of golf will be played in competition for the fifty trophies and prizes in the eight events. The trophies include the Association Championship for thirty-six holes, gross, the Association Handicap Championship for thirty-six holes net, the Choice Score Handicap Championship for thirty-six holes gross, the low gross Eighteen Hole Championship, the low net Eighteen Hole Handicap Championship, the Maturity Event limited to Fellows over 60 years of age, the Oldguard Championship limited to competition of past presidents, and the Kickers' Handicap. Other events and prizes will be announced at the first tee.

Dr. Homer K. Nicoll of Chicago is president and Dr. Charles Lukens of Toledo and Dr. John W. Powers of Milwaukee are vice-presidents of the American Medical Golfing Association, which was organized in 1915, by Dr. Will Walter, Dr. Wendell Phillips and Dr. Gene Lewis, and now totals 1,100 members representing every state in the union. The living past presidents include Dr. Wendell Phillips of New York, Dr. Thomas Hubbard of Toledo, Dr. Fred Bailey of St. Louis, Dr. Edward Martin of Philadelphia, Dr. Robert Moss of San Antonio, Dr. Charlton Wallace of New York, Dr. Will Walter of Chicago and Charlottesville, Va., Dr. James Eaves of San Francisco, Dr. Chester Brown of Danbury, Conn., Dr. Samuel Childs of Denver, Dr. W. D. Shelden of Rochester, Minn., Dr. Walter Schaller of San Francisco, Dr. Edwin Zabrisko of New York, Dr. Frank Kelly of Detroit and Dr. John Welsh Croskey, Philadelphia.

CLEVELAND COMMITTEE

The Cleveland Committee is under the chairmanship of Dr. John B. Morgan, 1301 Medical Arts Building, Cleveland, Ohio. He will be assisted by Drs. R. H. Birge, A. V. Boysen, E. F. Freedman, F. T. Gallagher, Secord Large, E. P. McNamee, J. J. Marek, Theodore Miller, U. V. Portman and M. A. Thomas.

The Mayfield Country Club of Cleveland is described by Chairman Morgan as "probably the finest course in the district, and certainly one of the most interesting. Many championships have been held on this course, and I am sure the visiting doctors will be delighted with it in every sense of the word. It has a most beautiful club house, and we can promise a merry nineteenth hole and a dinner fit for a champion."

APPLICATION FOR MEMBERSHIP

All male Fellows of the American Medical Association are eligible and cordially invited to become members of the A. M. G. A. Write the Executive Secretary, Bill Burns, 4421 Woodward Avenue, Detroit, for an application blank. Participants in the A. M. G. A. tournament are required to furnish their home club handicap, signed by the secretary. No handicap over 25 is allowed, except in the Kickers'. No trophy is awarded a Fellow who is absent from the annual dinner.

The twentieth tournament of the American Medical Golfing Association promises to be a happy affair, attended by some two hundred medical golfers from all parts of the United States.

REPORTS AND NOTICES OF MEETINGS

THE WILLIAM HARVEY SOCIETY

The William Harvey Society met in the Auditorium of the Beth Israel Hospital on April 13. Dr. Albert A. Epstein of New York City spoke on the "Disease of the Kidney in General Practice."

Dr. Epstein limited himself to a few of the more important processes that take place in kidney disease. He pointed out that the pathologist has helped to confuse the subject by describing the end products, while we are really interested in the processes by which these end results in the kidneys are obtained.

The question of albuminuria is an important one, though poorly understood. Albumin in the urine includes a variety of protein substances. The presence of these substances in the urine does not necessarily mean kidney disease; for example, it is normal to have an albuminuria after cold baths, during or after severe exercise, and in students at examination time. Albumin, in these cases, represents the passage of protein from the blood as a result of circulatory disturbances. Orthostatic albuminuria, certain cases of severe lordosis or any of various causes of such lordosis may be the cause of albumin appearing in the normal urine. None of these conditions represent kidney disease, nor do they lead to kidney disease.

The presence of red blood cells, white blood cells, and casts are of much more significance, though even they may not represent kidney disease, since they may arise from other parts of the urinary tract, as is the case with numerous calcium oxalate crystals which traumatize the ureters and cause an inflammation together with a mechanical effect on the tubules.

The clinical condition known as nephrosis presents a urinary picture in which there is the passage of varying amounts of albumin. This loss of protein may amount to as much as one or two ounces of albumin a day. The normal plasma protein varies from six to eight and one-half Gm. per cent and this figure may be lowered to two Gm. per cent in a long-continued albuminuria. The repeated withdrawal of ascitic fluid will lead to the same low plasma protein content.

The first important clinical effect of such albuminuria is edema. This is caused by the fact that the osmotic pressure of the protein of the plasma has been so reduced that the blood pressure forces an abnormal amount of fluid into the tissue spaces. The fluid balance in the tissue spaces is upset in this manner whenever the protein falls below five Gm. per cent, and the normal osmotic pressure which tends to hold the fluids in the vessels is no longer great enough to carry out its task. Furthermore, there is a reversal of the normal two-to-one albumin globulin ratio. This is because the albumin molecule is smaller than the globulin and passes through the kidney more easily, but the destruction of the tissue protoplasm also contributes considerably to this increase in globulin.

In albuminuria fats are mobilized in the blood and represent the lipoids that are closely associated with the cells that break down in the general tissue disintegration, a disintegration that is caused by the edema and poor nutrition of the body cells. Another effect of albuminuria is that it causes, through the undernutrition of the cells, a fall in the B.M.R. which in turn causes changes in the thyroid suggesting an exhaustion.

In these cases of heavy albumin loss where the entire clinical picture is due to the effects of this loss, the institution of the proper treatment will bring about a cure unless infection sets in. Such treatment takes from six months to four years and consists in replacing the deficit of protein by a heavy protein diet as well as the use of large doses of thyroid, up to 60 grains a day, in order to facilitate the use of the protein. The lipoid content of the blood is used as an index of how far the thyroid treatment can be carried, since the overactivity of the thyroid causes a proportional decrease in blood lipoids.

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The New England Roentgen Ray Society held its meeting on Friday, April 20, 1934, at 8:15 P.M. in the auditorium of the Beth Israel Hospital. Dr. Samuel A. Robins presided.

The first paper presented was by Dr. David Davis, who spoke on "Roentgenological Changes in the Heart after Total Thyroidectomy." The changes in the heart picture following total thyroidectomy depend in part on the condition of the heart before operation. A few patients with cardiac decompensation before operation have shown a decrease in the size of the heart shadow. Some have shown no change, probably due to a balancing of the effect of thyroidectomy with the improvement in the condition of the heart. Ordinarily, there is a slight increase in the size of the heart shadow after operation, this increase comes soon after the operation,

coincident with the lowered metabolic rate. Dr. Davis reported fifteen patients with congestive heart failure in whom total thyroidectomy was followed by an increase in the heart shadow size and by clinical improvement. The myxedema heart was briefly discussed.

A paper on "Protracted Irradiation in Carcinoma of the Larynx" was presented by Dr. Harry F. Friedman. He reported his results in the treatment of five cases of carcinoma of the larynx and pointed out that the intensity of irradiation in this disease should be influenced by the amount of calcification that is present in the laryngeal cartilages. Papillary types of carcinomata require the largest doses. The speaker showed slides demonstrating the site of irradiation and the subsequent skin reaction. He demonstrated two patients treated by x-ray with encouraging result. One patient has gone fourteen months without recurrence. Dr. Friedman described another of his patients who was irradiated every day from 9 A.M. to 5 P.M. for thirty-five days. Aphonia follows the treatment with gradual improvement. Tracheotomy has occasionally been necessary because of the reaction. There is little discomfort from the skin reactions with no apparent untoward effects upon the thyroid gland.

Dr. William S. Altman presented an interesting paper on "The Value of Lateral Views of the Rectum." It has been found from experience that the left lateral view gives the best results. The plates are best taken immediately after the sigmoid is filled and before the entire colon is filled, because loops of the latter may easily obscure lesions in the former. Dr. Altman showed lantern slides demonstrating lesions in the sigmoid on the lateral view which had been entirely concealed in A-P or P-A views. Lateral views have proved useful in the demonstration and diagnosis of lesions of the descending colon.

The subject of "Roentgenological Explorations of the Biliary Ducts with Lipiodol" was discussed by Dr. Louis Hermanson. The procedure has been reported only once by an observer in Argentina, and attempts are now being made at the Beth Israel Hospital to make it of practical use. Lipiodol is injected into the biliary ducts at the time of operation by means of a 14 gauge needle in quantities of 15 to 20 cc. There is no necessary modification of the operative procedure except that the drapes are sewed on rather than clipped. The picture thereby obtained is of considerable value to determine whether the common duct is to be explored, at least in the doubtful cases. X-rays may be taken at any desired interval after the operation as check-ups.

Dr. Leopold Brodny presented an interesting paper on Urethrography and illustrated it by means of lantern slides. Urethrography has been neglected largely because the urethra is so readily accessible to the urethroscope. The work started with Dr. Cunningham in Boston in 1910, but the posterior urethra was not demonstrated by urethrography until 1921. Any iodized oil may be used for the injection. After about 20 cc. of the fluid has been injected, the x-ray film is exposed, but care has to be taken that the fluid continues to be injected. If the injection is stopped at the time the exposure is made, the posterior urethra shuts down and will not appear in the film. The position of the patient is important for good pictures. Lantern slides showing urethrograms in the presence of prostatic hyperthrophy were projected on the screen. The splitting of the stream of injected material in the posterior urethra with hypertrophy of the middle lobe of the prostate was very clearly demonstrated. Urethrograms in patients with tabetic bladders show a characteristically funnel-shaped posterior urethra. The condition of the urethra after prostatectomy was illustrated. It was apparent that good functional results are not dependent exclusively on anatomical restoration. The internal sphincter is not essential for urinary continence. Urethrograms in the female were also demonstrated, showing anatomical variations, a case with a small diverticulum of the urethra, and a case with hypertrophy of the internal sphincter. The effect of straining on the position of bladder and urethra relative to the pelvis in cases with cystocele was shown very adequately by means of urethrograms.

The final paper was presented by Dr. Samuel A. Robins who spoke on "Cystography and the Prostate." Cystography was first used at the beginning of this century and shortly thereafter attempts were made at demonstrating enlargements of the prostate by injection of air into the bladder. Solutions of the halogens were first used in 1919, in 25 per cent solutions. In his experience, Dr. Robins has used 3 per cent solutions very satisfactorily. Various affections of the bladder were shown by means of lantern slides, including hypertrophy, sacculation, pouching, and elevation of the base of the bladder. The characteristic "derby-shaped" appearance of the bladder with elevated base due to prostatic hypertrophy and obstruction was illustrated by numerous examples. Distinct trabeculation was shown in some of the slides presented, as well as small defects from hypertrophy of the median bar of the prostate and defects from the small fibrotic prostate. Dr. Robins pointed out that by cystography it is impossible to make a diagnosis of carcinoma of the prostate.

Each of the papers was followed by a very brief discussion.

MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

The Massachusetts Association of Boards of Health met on May 3 at the University Club, Boston.

Dr. Henry D. Chadwick, Commissioner of the State Department of Public Health, was elected to membership on the Executive Committee. Mr. Donald G. Buckner was elected to fill the unexpired

term of secretary-treasurer, recently made vacant through the decease of Mr. George T. Lennon, who had served faithfully for many years.

Dr. Francis P. Denny, as the chairman of the committee to arrange for certain sections within the organization, reported that he had received requests for several sections as follows: Health Officers, 41; Laboratory, 25; Milk and Food Inspection, 25; Sanitary Engineer, 13; Epidemiology, 31; Public Health Nursing, 14.

The following papers were read: Mr. David A. Chapman, on "Air Pollution"; Prof. Philip Drinker on "The Cause and Prevention of Silicosis"; Prof. William F. Wells on "Air Bacteriology." Following the reading of these papers a general discussion was entered into by many of the members present. A rising vote of thanks was tendered to the gentlemen who read the papers.

JOHN J. MCGRATH, *Agent.*

FAULKNER HOSPITAL CLINICAL MEETING

On Thursday afternoon, May 3rd, at 5:00 P.M. the regular monthly clinical meeting of the Staff of the Faulkner Hospital was held at the hospital.

Two unusually interesting cases which had come to autopsy during the month were discussed.

In one case the outstanding features were pronounced anemia, ulcerations around and in the anus and peculiar degenerative lesions of the skin. At the time the patient first came under observation there was a marked leukopenia so that at first the case was thought to be one of agranulocytosis. During the patient's stay in the hospital, however, with the only specific treatment consisting in injections of liver extract, the leukocyte count rose from 650 to around 18,000. At autopsy it was found that the case was one of aleukemic leukemia.

In the other case the outstanding symptom was loss of weight with slight fever. X-ray pictures of the pelvic bones showed many lesions which suggested metastatic malignancy of the type in which there was increase in density in the shadow cast. Shortly before death this patient had considerable precordial distress and the pulse rate became markedly accelerated and irregular in rhythm. At autopsy there was no evidence of malignancy upon gross inspection and the bones which showed the increase in shadow were softer than usual. The pericardial cavity was filled with blood, but it was not evident from where the blood came. On microscopic examination, however, all the organs of the body were filled with an adenocarcinoma. There is still doubt in the mind of the pathologist in regard to where the tumor originated. The structure of the adenocarcinoma is consistent with origin in the biliary passages but evidence of tumor was present in the ovarian tissue, which is an unusual organ for metastasis from a tumor originating in the liver.

Dr. William W. Howell then gave a short discussion on "The Methods of Handling Difficult Problems in Infants and Children," and in a very amusing manner told of some of his personal experiences in which common sense and judgment seemed to have been lost sight of by physicians and parents in the handling of the individual problem. Although not in any way trying to detract from the value of various diagnostic and therapeutic procedures which have been recently introduced into the practice of pediatrics, he sounded a note of warning that in some instances too many of these procedures may be tried upon the individual patient and too often there is a lack of judgment on the part of the physician in regard to how many of these different procedures should be used. He emphasized the fact that up to the present time a great majority of children seem to grow up into healthy adults without worrying over a slight loss of weight for a day or so after birth or feeling that dehydration should be guarded against. One gathered also from listening to him that possibly the great majority of babies would do as well without the curiosity of the physician being satisfied in regard to the thymus gland by an x-ray study shortly after birth.

The final clinical meeting of the season will be held on Thursday, June 7th, at 5:00 P.M., at the hospital. In addition to the usual clinical pathological conference Dr. Edward L. Young, Jr., who was scheduled to speak at the April meeting, but was unable to do so, will discuss "Some of the Difficulties of Gallbladder Diagnosis." All physicians who are interested are cordially invited.

THE CARNEY HOSPITAL CLINICAL MEETINGS

On Wednesday morning, April 25, the regular clinical monthly meeting was held at the hospital. About forty visiting physicians and members of the Staff were present.

Dr. A. McK. Fraser did a cholecystectomy and drainage of common bile duct.

Dr. Harold Lee did an open operation for fractures of both bones of forearm.

The Dry Clinic, which was well attended, started at 10 A.M., and lasted until 12 o'clock. During this time three members of the Staff gave short talks.

Dr. A. Leo Brett discussed certain types of foot strain associated with backache, relieved by foot plates, and demonstrated various forms of plates.

Dr. L. E. Phaneuf discussed avoidable accidents in obstetrics.

Dr. A. McK. Fraser discussed care of patients before operations for gallbladder disease.

The next clinical meeting will be held at the hospital on Wednesday, the 23rd of May, and at that meeting various operations will be performed by members of the Staff.

A Dry Clinic will be given by Drs. Louis F. Curran, Daniel J. Hogan, and Norman A. Welch, of the Medical Department. The subject presented will be "Electrocardiography."

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

ANNUAL MEETING

The Copley Plaza, Wednesday, May 23, 1934.
Business Meeting 6:30 P.M.
Dinner at 7 P.M. sharp. $2.50 per plate.
Election of Officers.

Speakers:

1. Dr. Joel E. Goldthwait, "Chronic Invalidism due to Joint Pathology."
2. Dr. Howard Thompson, "Medical Aspects of the Subject."
3. Dr. Carl Watson, "The Subject from the Osteopathic Point of View."

CADIS PHIPPS, M.D., *President.*
WM. PEARCE COUES, M.D., *Secretary.*

AMERICAN PSYCHIATRIC ASSOCIATION

The ninetieth annual meeting of the American Psychiatric Association will be held at the Hotel Waldorf-Astoria, New York City, May 28, 29, 30, 31, and June 1, 1934.

The program which includes addresses by eminent psychiatrists representing the several sections of the country is of especial interest.

There will be a scientific exhibit, motion pictures of hospital activities, and a commercial exhibit. Special rates are allowed by railroads. To secure these reduced rates, those who attend should obtain a certificate (not a receipt) for each ticket. One-way ticket should be bought to New York and on presentation of the certificate at headquarters registration, the return ticket can be purchased at one-third fare.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

May 18—The New England Roentgen Ray Society will meet at the Boston Art Club, Boston, at 8 P.M. The Annual Dinner will be held at the Boston Art Club at 6:45 P.M.

May 19—The New England Pediatric Society will meet at New Haven, Conn., at 2:30 D.S.T. Information may be obtained from the Secretary, Dr. Gerald Hoeffel, 319 Longwood Avenue, Boston.

May 21, 24, 25—New England Heart Association. See page 1041, issue of May 10.

May 23—Massachusetts Society of Examining Physicians. See notice above.

May 23—The Carney Hospital Clinical Meeting. See page 1093.

May 26, 27, 28, and 29—The American Association on Mental Deficiency. Details may be obtained from the Secretary, Dr. Groves B. Smith, Godfrey, Illinois.

May 28 - June 1—American Psychiatric Association. See notice above.

June 7—Faulkner Hospital Clinical Meeting. See page 1093.

June 7, 8, 9—Annual Meeting of the American Association for the Study of Goiter will be held in Cleveland, Ohio. For details of the program apply to Dr. J. R. Yung, Terre Haute, Ind.

June 11—American Medical Golfing Association. See page 1090.

June 12—American Heart Association will meet at 9:30 A.M. at the Cleveland Hotel, Cleveland, Ohio.

July 24-31—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

August 18 - September 30—Medical Study Trip to Hungary. See page 975, issue of May 10.

September 3-6—American Public Health Association, at Pasadena, California. Dr. J. D. Dunshee, Chairman, Local Committee on Arrangements.

September 4, 5, 6—International Union Against Tuberculosis. For information address the National Tuberculosis Association, 450 Seventh Avenue, New York City.

BOOKS RECEIVED FOR REVIEW

Traité de Physiologie Normale et Pathologique. Tome V. Respiration. Par M. M. Léon Binet, et al. 474 pp. Paris: Masson et Cie. 80 fr.

The Clinical Management of Horseshoe Kidney. By Robert Gutierrez. 143 pp. New York: Paul B. Hoeber, Inc. $3.00.

International Clinics. Volume I. Forty-Fourth Series, 1934. Edited by Louis Hamman. 320 pp. Philadelphia: J. B. Lippincott Company.

Transactions of the American Gynecological Society. Volume 58, for the year 1933. Edited by Otto H. Schwarz. 318 pp. St. Louis: C. V. Mosby Company.

The Deserted Village. No. 2. 63 pp. New York: The Chemical Foundation.

Chinese Medicine. By William R. Morse. 135 pp. New York: Paul B. Hoeber, Inc. $2.50.

Allergy in General Practice. By Samuel M. Feinberg. 339 pp. Philadelphia: Lea & Febiger. $4.50.

Manometric Methods as Applied to the Measurement of Cell Respiration and Other Processes. By Malcolm Dixon. 122 pp. New York: The Macmillan Company. $1.75.

13 Jahre Wissenschaftlicher Medizin im Nord-Kaukasus. 1920-1933. Edited by I. L. Benkowitsch. 244 pp. Rostow am Don: Verlag "Sewerny Kawkas."

Aids to Qualitative Inorganic Analysis. By R. G. Austin. Students Aids Series. 204 pp. Baltimore: William Wood and Company. $1.50.

Aids to Pathological Technique. By David H. Haler. Students Aids Series. 187 pp. Baltimore: William Wood and Company. $1.50.

The Pocket Anatomy. By C. H. Fagge. Ninth Edition. Students Aids Series. 333 pp. Baltimore: William Wood and Company. $2.00.

The Harvey Lectures. Delivered under the auspices of the Harvey Society of New York. 1932-1933. Series XXVIII. 233 pp. Baltimore: The Williams & Wilkins Company.

BOOK REVIEW

Aids to Neurology. By E. A. Blake Pritchard. 376 pp. Baltimore: William Wood and Company. $2.00.

This is a students' review book, well written and accurately describing the main signs and symptoms of nervous diseases. It should be useful as a brief summary of the subject to both the student and the practitioner.